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SOME REFLECTIONS ON THE DIGESTIVE PROCESS*

CALDWELL LECTURE, 1934

By WALTER B. CANNON

George Higginson Professor of Physiology, Harvard Medical School

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INTRODUCTORY REMARKS

By PERCY BROWN

Once again the American Roentgen Ray Society is met to listen to its Caldwell Lecture, of its annual scientific program the principal feature, founded fourteen years ago in memory of the life and works of Eugene Wilson Caldwell, its former President and Dean of American Roentgenology.

For the ears of the younger of you, be it reiterated that Doctor Caldwell was trained as an electrical engineer. Coming east from the University of Kansas to enter into the practice of his profession, the moment of Professor Röntgen's classic Preliminary Communication found him occupied with telephonic work as construction engineer for the New York Telephone Company, having recently completed a series of experiments in submarine wireless telephonic signalling for the United States Lighthouse Establishment.

The momentous reports from the Physical Institute of the University of Würzburg attracted Caldwell as they attracted scientists of his type the world over, but it was not until many months later, that he, a young engineer in a large community without resources or influence, was able to divorce the occupation of his necessity and the quasi-vocation of his desire. The success of such transition required (in the order in which he found them necessary) some apparatus, a laboratory, a knowledge of anatomy, a medical education and—material, in other words, patients. The latter designation always, in later years, threw Caldwell into profound depression; to him it connoted the nice amenities of clinical contacts that had no appeal for him.

Caldwell's first apparatus was acquired from a commercial photographer who had failed in an attempt to exploit the roentgen rays as a popular diversion; his "laboratory," a mere cubicle of an office where the desultory and primitive x-ray efforts of 1898 were, in his case, sustained by occasional opportunities in engineering consultation and design; his anatomy, by a year's special course at Columbia University with evening dissection. In 1901 came the realization that the future of this work required of him, or of anyone else, a full medical education; with this in view, Caldwell entered the University and Bellevue Hospital Medical College of New York University, and was graduated therefrom in 1905, just thirteen years from the date of his Bachelor's degree in Science from the University of Kansas. By now, "material," in the form of patients, was his in plenty; its augmentation necessitated repeated acquisition of laboratory space for his own practice, aside from his work at the Gibbs Laboratory of his medical school, which was created for him.

The course and accomplishments of Caldwell's professional life are known in detail to us who were his contemporaries; for others, it is

* The fifteenth Caldwell Lecture, delivered at the Thirty-fifth Annual Meeting of the American Roentgen Ray Society, Pittsburgh, Pa., Sept. 25–28, 1934.

well recorded in the literature. A few facts have been recited to show that his transition into the field of Medicine had no effect on the depth of his dye as a laboratory-trained physicist, and that, once in Medicine, it was experimental investigation that dominated his thoughts and often made him orally wistful. "I think I should be fairly happy in my work," he once remarked, "if it were not for patients. They take time, and there is so much, so very much, to be done." Caldwell was indeed, to quote Molière, "*Le médecin malgré lui*"—his heart was never far from the tables of the physical and engineering laboratories.

Caldwell's ultimate medical education, however, spread before his inventive genius an array of entities in pathology that offered urgent problems in the practical application of this wondrous new manifestation of pure science. Always, the degree of his creative effort in engineering was measured by the complexity of the quest confronting him, whereof the solution marked a forward step in diagnosis. Often his accomplishments to this end, due to his extraordinary vision, were so far ahead of the general development of roentgenologic science at that moment that the practical reasons for them were not clear to a less elastic imagination. Witness, by way of example, Caldwell's system of valve-tube rectification that was a demonstrable actuality in his laboratory long before the decline of the cold-cathode focus-tube; again, we are perhaps in the habit of considering his stereoscopic fluoroscope, which he just lived to see completed, to be entirely the outcome of a war-time suggestion; in point of fact, Caldwell had worked out the whole principle, with minor variation, seventeen years previously, in a practical apparatus that was described in detail in the *Electrical Review* of 1901.

No one, therefore, could have been more keenly sensitive than he to all important steps in the forward progress of experimental research in the various fields of applied science, especially that of diagnostic roentgenology. The prophetic vision that seemed to guide to fruition his undertakings in the technics of this subject likewise enabled him to evaluate with unerring acumen the accomplishments of his contemporaries in their bearing on the future development of radiology in general.

Not far from the year 1905, in discussing the research strength of some of the great American medical schools in contrast with the laboratories of Europe, Caldwell said to a Harvard friend: "The exceedingly important investigations of your physiologist, Doctor Cannon, clearly indicate the extent to which the development of our work [i.e., roentgenology] depends on carefully made and accurately recorded observation. The effect of this research is certain to be far-reaching."

To what research did Caldwell refer? This was its inception:

In early May of 1897 (and the date is memorable for other reasons than that it was but sixteen months after the discovery of the roentgen rays) there were presented, to the fourth special meeting of the American Physiological Society by Dr. W. T. Porter for Professor Henry P. Bowditch, two reports "on the results of experiments performed in the laboratory of the Harvard Medical School by Messrs. W. B. Cannon and A. Moser, medical students, on the movements of the alimentary canal as studied by means of the x-rays and a fluorescent screen." Their titles were: "The movement of the food in deglutition" by Mr. W. B. Cannon and Mr. A. Moser and "The movements of the stomach studied by means of the roentgen rays" by Mr. W. B. Cannon. The summaries of these reports appeared in *Science*, June 11, 1897, and in the Proceedings of the American Physiological Society at its tenth annual meeting in late December of 1897, with additional record of work done, and the full paper, from the pen of Mr. Cannon, in the first volume of the *American Journal of Physiology*, May, 1898, under almost exactly the title of the second of the preliminary reports.

This contribution of a keenly-observing medical student constitutes, ladies and gentlemen, the central foundation-stone that solidly underlies the enduring structure of alimentary diagnosis through the agency of modern roentgenology. From its modest origin within the walls of the physiological laboratory, through the mediumship of the common cat, one of many of God's creatures thus ennobled, has developed an instrument for the relief of human sorrow and suffering, and the prolongation of human life, than which there is none more potent.

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On our way to lecture, as we passed the laboratory door, occasionally his classmates would see Cannon seated at his work therein. With an intuition that even the medical student at intervals possesses, we realized that, while we

were flogging the slow-moving flanks of our mental oxen, here, indeed, was one of us perched between the wings of Pegasus; a Bellerophon off on a quest that we could not then understand, although the maturer scientific world was even then learning that it was no Chimaera! It is without chagrin, and only in pride, that we have watched his flight.

To this quest, Doctor Cannon gave his close attention for twelve years. The thorough manner of its undertaking will be realized if the gross subdivisions of his initial communication be even quickly recited; they are, aside from an exposition of the literature and an explanation of his method, (*c*) the anatomy of the stomach and its relations to the shadow; (*d*) the normal movements of the stomach as to the pyloric part, as to the pyloric sphincter, and as to the activity of the cardiac portion; (*e*) the movements of the stomach in vomiting; (*f*) the effect of the movement of the stomach on the food; (*g*) salivary digestion in the stomach; (*h*) the inhibition of the stomach movements during emotion. Further work in elaboration of these matters, and kindred observations of the more distal alimentary tract, made up this long period of investigation on the motor phenomena of digestion, and the findings thus attained resulted in Doctor Cannon's monograph of 1911—"The Mechanical Factors of Digestion." It is this group of his studies that, of course, holds the greatest interest for the roentgenologist, but as students of the progress of physiology and his influence on it, we can take equal pleasure in reviewing the course of Doctor Cannon's work in a new field which he embraced in 1912, after demonstrating during the same year, in valediction to his previous labors, the physical basis of the sensation of hunger.

For twenty years, with one notable period of interruption, he has scrutinized the function of the autonomic nervous system under the supportive or reinforcing influence of certain endocrine tissue, notably the adrenal medulla. Based on this prolonged study is his volume, "Bodily Changes in Pain, Hunger, Fear and Rage."

The "period of interruption" in no way interrupted Doctor Cannon's power for human beneficence through his investigative genius. It was his War period—a long War period in France and England, that gave opportunity for his versatility at Camiers, at Béthune, in London, in Paris, at Dijon. The problems before him were those of surgical shock and hemorrhage, of the physical effects of gas-warfare, of the effect of cold on blood-pressure, beside the many lesser matters arising daily in his various laboratories. His results have enriched the scientific literature of the Great War, and by his energy have been saved countless lives.

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For us radiologists be the lessons of his earlier teaching, however. As we sit before our fluorescent screen, perhaps at our side a surgeon whose fingers are itching, may we resist the impulsion to seek an organic cause for every variation we see in alimentary behavior. It is for us to remember that the temporal body physiologic, as well as the ecclesiastical body politic, is ruled by Can(n)on Law!

No more need be said, therefore, if indeed have been necessary these feeble but prideful words of a medical classmate, to introduce to any lover of roentgenology—Dr. Walter B. Cannon, George Higginson Professor of Physiology at Harvard University.

CALDWELL LECTURE

WE MEET together this evening to pay tribute to the memory of one of the outstanding American pioneers in the field which this Society cultivates. It was not my privilege to know Dr. Caldwell personally, but from friends who knew him well I have rejoiced to learn of his many high qualities as a man and as a contributor to the science of roentgenology. His enthusiastic devotion to his chosen work, his ingenuity, his pursuit of the ideal of perfection were matched by equally admirable traits of character as displayed in loyal friendship, in good cheer, and in the bravery and fortitude with which he continued his labors during the painful last period of his lamentably shortened career. Such spirits we do well to commemorate, for they disclose to us the heroic way in which life may be lived when fate lays upon us a heavy hand.

Dr. Caldwell died a martyr to his services to the progress of medicine. In the early days the pioneers in roentgenology had no inkling of the dangers to which they were subjected. Well do I recall the sage advice of Professor Henry P. Bowditch, when, in the spring of 1897, I showed him

an area on my hand from which the skin, exposed repeatedly to the strange new rays, had come away in successive layers. He remarked that we were employing a quite novel agent which might have very dangerous properties and that we ought to be cautious until we learned more about its nature. At the time I was an eager first-year medical student, not pleased with being checked in my purposes. In order to avoid further harm I surrounded the tube with a metal box, leaving a small aperture at the top through which the rays could pass. The suggestion had been offered that the burns might be due to the brush discharge of the high tension current; you will be amused to learn that as a special precaution I grounded the metal box by a wire connection attached to the water pipes! Thus quite early I was protected. Consequently today only few scars remain, although during a decade, I often sat for hours together watching with the fluoroscope the processes of digestion in different parts of the alimentary canal.

Nearly thirty-eight years have passed since peristalsis in the esophagus of a goose was first demonstrated at the Harvard Medical School to members of the American Physiological Society—late in December, 1896. Some of the observations on the movements of the esophagus and the stomach have been published for thirty-six years, and more than a quarter-century ago practically all of my researches on the digestive tract ended. In October, 1913, I had the privilege of telling to this Society the story of the early use of the roentgen ray in the study of the alimentary canal.[13] During the period which has intervened, I have been concerned with various other aspects of physiology, so that the interest in digestion has gradually faded into the background. And yet, when the invitation came to speak to you again, I was tempted to return to a reconsideration of some of my investigations as a beginner in physiology. Only rarely does an investigator have the opportunity to look back through three decades to his primary efforts and their fruits, and to survey them with a fairly critical detachment. It has seemed to me that you as roentgenologists might be willing to undertake with me an evaluation of a few of the results of the initial employment of the roentgen ray on the digestive canal, and an estimation of work which still remains to be done.

Obviously the testimony which required a volume[12] for its exposition cannot be surveyed in a brief lecture. In selecting, therefore, certain topics to consider, I propose that we choose those over which there has been discussion, because they are likely to prove most suggestive for further study. Let us take up these topics one by one.

The Course of Gastric Peristalsis. In 1896 Professor Bowditch told his students that the butchers knew as much as the physiologists about the motions of the stomach, because the belly must be opened to see the waves and that operation might induce abnormal conditions. The roentgen rays, first used to examine the digesting stomach in April, 1897, supplied a standard by which the correctness of other modes of study could be judged. At that time the view was prevalent that the stomach contracts in two phases: the first, a peristaltic constriction which, starting near the cardia, ends in a strong contraction of the "transverse band" and divides the gastric cavity into two parts, the vestibule (or pars pylorica) and the fundus; and the second, a systolic contraction of the vestibule as a whole. That view was based on the inferences which Beaumont[6] drew from the motions and the encountered resistances of a thermometer which he introduced through a gastric fistula in his famous patient, Alexis St. Martin; and also on the observations of Hofmeister and Schütz[28] on the dog's stomach, which was excised and kept in a moist chamber at body temperature. The conditions in both cases were either obscure or abnormal. Against the notion of gastric peristalsis which was thus based I protested, stating that many observations on a variety of animals had not revealed the obliterating constriction at the sharp turn into the pyloric portion, and that the

peristaltic waves progressed even to the pylorus.[2 (p.53)] This description was in accord with the testimony of Kästle, Rieder and Rosenthal[30] and also of Carman,[18] regarding the process in man. To be sure, I did report waves which "at times almost totally obliterated the lumen" as they moved into the pyloric half of the vestibule, and my tracings showed shadows which can be explained only by a systole of that region,[8] but the emphasis was certainly on the phenomenon of a smoothly advancing peristaltic ring.

The careful studies by Alvarez,[1(p. 111)] with electrical, mechanical and photographic recording devices, leave no doubt that more or less of the pyloric portion of the stomach may manifest a true systole. Apparently conduction affecting the circular fibers becomes so rapid as the pylorus is approached that the progressive wave disappears in the diffuse contraction. Studies on man, dog and rabbit by M'Crea, M'Swiney, Morison and Stopford[38] have also shown, in addition to simple peristalsis, a concentric shortening of the muscles in the pyloric half of the vestibule. And in cats Borchers[7] noted that although the peristaltic type of activity usually prevails, it is interrupted at times by the occurrences of the systolic type. Apparently the extent of the region over which the systole reaches may vary, for occasionally the deepening at the "transverse band" has been seen, with attendant compression of all the content of the vestibule. In addition, Alvarez and Zimmermann,[3] Wheelon and Thomas,[48] and Klein[31] have all described conditions in which the movements in one part of the stomach were not coördinated with those in another part. To be sure, in these studies the observations were made on animals with the abdomen opened and with other unnatural interferences, but, even so, they indicate that the gastric musculature, instead of performing always in a routine manner, plays its rôle in different ways in different times and circumstances.

The question properly raised by these observations is whether the varieties of movements of the stomach are regularly associated with other phenomena. Does the systole of the pyloric region indicate discharge through the pylorus, as Borchers[7] surmised? If so, why does it occur sometimes and not at other times? Are the isolated activities which are seen in different parts of the exposed stomach observable in the diseased organ? Are there types of peristalsis peculiar to lesions in one part or another of the organ? Are there characteristic behavior patterns of the stomach associated with the ingestion of different substances, as Todd[47] has claimed? Careful observations, made not only after drinking a "barium meal," but after eating normal meals composed of various foods, might disclose interesting answers to some of these questions.

The Passage of Different Foodstuffs from the Stomach. Thirty-two years ago I reported[9] that when salmon is fed it leaves the stomach later and more slowly than bread and milk. The hint given by this observation was accepted, and a research was undertaken to learn whether the three different foodstuffs have characteristic rates of discharge into the intestine. Carbohydrates, proteins and fats were fed in uniform amounts, with fairly uniform consistency, and with a uniform admixture of bismuth subnitrate. The speed of the passage from the stomach was judged from measurements of the aggregate length of shadows of food in the small intestine which were traced at regular intervals[10]—a method later validated by Magnus,[39] and others. The method was admittedly subject to errors, but the claim was made that these errors, especially in the early stages of digestion and absorption, may be regarded as comparatively slight in relation to the large differences of intestinal content when the three foodstuffs are separately fed.

The rapid discharge of carbohydrate food from the stomach and the slow discharge of fat, disclosed by the method, corresponded to some earlier observations in which, however, careful comparisons were not made.[12(p.87)] The intermediate rate of discharge of protein had not been reported.

It is of interest that the results obtained by measuring intestinal shadows have since been confirmed by studies on human beings. Wulach[49] fed individuals having a normal digestive tract meals which were predominately carbohydrate, protein or fat. Care was taken that the amount, the consistency, the temperature and the salt content of the foods were approximately the same, and that the meals were fed at about the same time on different days—8:00 to 8:30 in the morning. Examination with the roentgen rays showed that the stomach emptied itself of carbohydrate food in 2.5 to 3.5 hours, or protein food in five to six hours and of the meal of fat in seven to 8.5 hours. Further testimony leading to the same conclusion was offered by Demuth[21] who, though he included tests on patients with gastric disorders, together with tests on normal persons, found that when the three foodstuffs were separately fed (300 grams of each), protein remained in the stomach approximately 25 to 45 per cent longer than carbohydrate, and fat 35 to 55 per cent longer.

The most recent study of the rate of gastric evacuation is that by Gianturco[23] who used the admirable technique of cinematographic roentgenography devised by Alvarez. He found that when different foods were fed in uniform amounts to fasting cats, starch (30 per cent), mashed potatoes or crackers and milk had left the stomach in four or five hours; coarsely ground meat in about nine hours; and olive oil in about twenty hours. These observations are quite in accord with the results reported by me in 1904. Gianturco has emphasized, however, the importance of the *consistency* of protein food in determining the rate of gastric discharge; he noted, for example, that finely ground liver and muscle went out with carbohydrate speed. It would be interesting to know how these preparations compared with the coarsely ground meat, for in 1909, Hedblom and I[2] reported that when a thin gruel of shredded meat is fed, it is passed onward from the stomach much faster than when the meat has the standard, thick, mushy consistency. Apart from the comment on the influence of the physical state of the food, the testimony of Gianturco is, in the main, in accord with that given by other investigators.

The chief criticism which I should make of all these observations, my own included, is that the factor of consistency was only roughly determined. In my research the carbohydrate and protein, after being finely broken or mashed, was moistened until it had, "as nearly as could be judged by the eye and by manipulation, the uniform consistency of thick mush." Moreover, the fat which was fed was similarly judged, and the effect of change from room- to body-temperature was not taken into consideration. It would be an important enterprise, I believe, to repeat on human beings the studies made on lower animals, with proper attention to the amount and especially to the consistency of the standard meal, which should obviously *not be fluid*.

I have spent, perhaps, too much time in discussing the rate of discharge of food from the stomach. The topic has, however, practical bearings. If for any reason the work of the stomach should be spared, the feeding of carbohydrate would clearly be a preferable mode of introducing carbonaceous food into the body as contrasted with feeding fat. On the other hand, if it should be desirable to keep food for a long time in the stomach and let it out slowly into the small intestine, a large admixture of fat would be indicated. In this connection it is interesting to note that when Nansen and Johansen[42] were forced to spend the winter in Franz Josef Land they crawled from their rude hut only twice, at times only once, a day and ate their fill of meat and fat, the combination which leaves the stomach most slowly.

The Control of the Pyloric Sphincter. I now take up the topic which, in the recent history of the physiology of digestion, has roused more discussion than any other—the way in which the pylorus acts. The word "pylorus" means watcher or guardian

of the gate. "Janitor justus" was another name given to the sphincter by the ancients. And when one sees, as Murphy and I did in 1906,[16] the pylorus holding against gastric peristalsis for many hours and not permitting the stomach contents to be driven onward into a damaged intestine, the implication in the old names seems justified.

There are observers who have made statements which, as I understand them, would indicate that the well-defined muscle at the exit of the stomach has no function whatever. Thus Cole,[20] McClure, Reynolds and Schwartz,[37] Wheelon and Thomas,[48] and Klein,[32] have declared that there is a relaxation at the approach of every peristaltic wave, i.e., the pyloric canal is merely a passage-way, rhythmically ready to be sluiced by the rhythmically expressed content of the vestibule. It is biologically anomalous to find throughout the vertebrate series a sphincter muscle between the stomach and small intestine and to assume that it performs no service. And not all observers agree with those mentioned above. In my report on gastric movements in 1898,[8] I noted that in the cat the chyme emerged from the stomach *occasionally*, at irregular intervals. In studies made on his patients Case also found that the pylorus opens at variable periods just as I had stated; his words were, "In fluoroscopic observations I have repeatedly seen (in man) the behavior of the pylorus as described by Cannon, the opening of the pylorus occurring irregularly."[19] Kaestle,[29] making use of serial roentgenograms, came to the same conclusion, and Groedel[24] likewise has testified that in man not every contraction of the antrum drives material into the duodenum. Another view has recently been advanced by Gianturco.[23] His records he interprets as showing that the pylorus contracts when a gastric wave reaches it, and relaxes with the succeeding wave of relaxation, but that the duodenal movements are irregular in time and intensity. Since food can enter the intestine only when the pylorus and the duodenum are simultaneously open, he is driven to the conclusion that the action of the duodenal muscle must govern the emptying of the stomach. This view, again, seems to me to leave unexplained the "why" of the existence of the pyloric sphincter—a biological question, which because of the innumerable correlations between structure and function, we are justified in asking.

Struck by the remarkable difference between the speed with which carbohydrate and protein foods are discharged from the stomach, I offered, in 1907, the theory that the pylorus may normally be controlled by the hydrochloric acid of the gastric juice.[11] The assumption was made that when acid, imported from the fundus, has given to the food on the gastric side of the sphincter an acid reaction (recall that acid is not secreted in the pyloric portion of the stomach), that would signal the opening of the pylorus, and when the acid chyme is pushed into the duodenum its presence there would overcome the influence from the stomach and close the pylorus. At the same time, however, the acid in the duodenal contents would stimulate the flow of alkaline bile and pancreatic juice, which, by neutralizing the acid, would in time allow the passage of more acid chyme from the stomach, and so on until the stomach would be emptied. On this theory the different rates of emergence of starchy and protein foods from the stomach might be explained: both foodstuffs are known to stimulate gastric secretion, but the proteins, uniting with the secreted acid, would delay the development of an acid reaction and therefore would be passed out slowly, whereas, the carbohydrates, not uniting with the acid, would be passed out rapidly. The slow emergence of fats could be explained because, when they are fed, they check the flow of gastric juice, they excite slow and weak peristaltic waves, and also, according to Lintwarew,[33] when in the duodenum they are able to inhibit gastric evacuation.

It was a very pretty theory! Not only did it reasonably account for the differen-

tial retention of the three foodstuffs in the stomach, it also explained how *time* would be given for gastric digestion (until acid reached the pylorus from the glands in the fundus), how continuation of the digestive process by pancreatic juice and bile would be assured (for acid in the duodenum stimulates their flow), and how again time would be provided for mixture of the chyme with these important fluids (because the acid chyme holds the pylorus closed and thus checks further exit until the acid is neutralized). Moreover, feeding carbohydrate moistened with sodium bicarbonate caused an *initial* delay in the discharge from the stomach, and feeding protein as acid protein resulted in its rapid emergence. And if the pancreatic and bile ducts were tied, so that the acid chyme in the duodenum could not be readily neutralized, the stomach was shown to empty slowly.

Huxley once said that a tragedy of science is the killing of a beautiful theory by ugly facts. And that may be the fate of the theory I have just outlined. Some of the "ugly facts" which have recently been urged against it were, to be sure, frankly recognized when the theory was first propounded, twenty-seven years ago. For example, it is said that rapid discharge of water and egg-white does not accord with an acid control of the pylorus; but, as was pointed out at the time, they are peculiar, —they are fluid, they are not masticated with relish and swallowed slowly as are ordinary foods, and they do not excite, or excite only feebly, a secretion of gastric juice. The fact that ordinary foods evoke gastric secretion is not contradicted by the failure of water to do so, and similarly the idea that ordinary foods are treated in a typical manner at the pylorus should not be rendered dubious by the quick passage of water through the stomach. Again, the condition of *achylia gastrica* has been cited in opposition to the theory; but that condition is pathological and, as I pointed out long ago, such a state requires its special study to explain the facts—the pylorus, for instance, may be abnormally patent, as a part of the disease. Abnormal functioning of other parts of the body is not cited as nullifying the evidence as to the normal functioning of those parts. Why is it done here? In this same category of minor criticisms I would place the objection that other stimuli than acid affect the closing of the pyloric sphincter. The basis for that comment was largely furnished by observations made in the Harvard Laboratory! Hard, lumpy material in the stomach, damage to the duodenum, inflammation in the proximal colon—all these states, we found, delay the emptying of the stomach, without stopping gastric peristalsis. It was inferred, therefore, that they exerted their influence on the pylorus.

The "ugly facts" thus far mentioned seem to me not really damaging to the theory of an acid control of the pyloric sphincter. But there are others which are! In offering the theory I counted heavily on the testimony of Tobler[46] and of London and his collaborators[34] that the chyme discharged from the stomach is "strongly acid," on the reports of Hirsch[27] and Serdjukow[45] and Tobler[46] that acid introduced into the duodenum inhibits gastric discharge. And if I had brought forward the theory at a later date I could have cited the testimony of Morse[41a] and of Shay and Gershon-Cohen[44a] that acid added to water retards its passage onward—an effect on the pylorus from the duodenum—and also I could have pointed to the observations of Cowie and Lyon[20a] that in children the emptying of the stomach is retarded when the food (skim milk) is acidulated. It should be emphasized, too, that the theory was based largely on the evidence advanced by Bayliss and Starling[5] that acid excites the flow of alkaline pancreatic juice and bile by producing secretin. Opposed to this support for the theory are the studies of Luckhardt, Phillips and Carlson[38] who found that food material (fluid, to be sure), passed through the pylorus, often contains no free acid and rarely much "total" acid; and, in addition, the researches by McClure, Reynolds and Schwartz[37] and Bársony and Egan,[4] who

were unable to demonstrate any influence of acid, brought to the duodenum through a duodenal tube, in checking the outgo from the stomach. Such observations strike fundamentally at the view that the coördination of gastric and intestinal digestion is managed by the action of acid chyme on the pyloric sphincter. May I point out, however, that these observations would require, if proved correct, an important revision of our understanding of a significant step in the connected series of steps which constitute the digestive process. We now regard the repeated discharge of acid chyme into the duodenum as the cause of the production of secretin. This continues the flow of pancreatic juice and bile, until the last of the food has left the stomach. If the chyme is not acid as it passes through the pylorus it obviously cannot serve as an acid stimulus for the production of secretin. Furthermore, it should be noted, pepsin requires an acid reaction for its efficacy, with an optimum pH for different proteins that has been determined as ranging from 1.5 and 3.5—i.e., a pronounced degree of acidity. Chyme forced through the stomach without being acidified would not only be deprived of the chance of digestion by pepsin but also, according to present views, would not carry with it the condition for its digestion by trypsin. Thus, the arguments against the acid theory of pyloric control upset the physiological applecart in a quite thorough manner.

In closing this rather long discussion of the mechanism of interaction between gastric and intestinal processes, may I point out again that originally my intent was to explain the differential rate of discharge of different foodstuffs from the stomach. No other explanation than that of the effect of acid on the two sides of the pylorus has been offered to account for the phenomenon. Moreover, in spite of years of criticism, no one has taken the trouble to repeat exactly either my observations on the emptying of the stomach or the factors affecting the action of acid chyme that might influence the speed of emptying. Perhaps it is appropriate to suggest that an opportunity for research lies here—an opportunity which might show that my view is quite incorrect; indeed, that our notions of gastrointestinal physiology are incorrect.

The Question of "Tonus" of the Alimentary Canal. In discussing the rapid passage of water and egg-white from the stomach, I pointed out that they are not chewed and swallowed with a satisfaction of appetite, and they are not stimulators of gastric secretion, as are ordinary foods. A possible implication here is that when ordinary foods are chewed and swallowed the process not only induces a secretion of "psychic" or "appetite" juice from the salivary and gastric glands, as Pavloff[43] showed, but that it establishes a state of moderate tension or "tonus" in the gastric musculature, including the pyloric sphincter. This tonic state is the condition for rhythmic action. The idea that there might be a psychic "tonus" I suggested many years ago (cf. 12, p. 200), on the evidence obtained when the vagus nerves were severed before or after the start of digestion. When the nerves were cut before digestion had commenced, the gastric wall remained flabby and peristalsis did not begin for hours, and even when it began the waves were extraordinarily shallow. After digestion was well initiated, however, the vagi could be sectioned without altering either the nature of the peristaltic waves or the rate at which the stomach emptied itself. It seemed clear that the gastric muscle, as well as the gastric glands, received an influence by way of vagal paths that was important for the normal course of the digestive process. Quite recently evidence confirming that conclusion has been published by Meek and Herrin.[41]

The inference that there is a psychic tonus has been supported by a number of investigators, notably Alvarez.[2] One of his patients had a large ventral hernia, and the bowel was so thinly covered by peritoneum and skin that its contractions could be easily seen. When a nurse brought food, a

peristaltic wave was observed rushing over the bowel, followed by active kneading movements. Another patient described by Alvarez had an incompetent anal spincter and was compelled to be extremely careful about seeing, smelling or even thinking about the pleasures of the palate because of the stimulating psychic effects on peristalsis, and possible disastrous consequences therefrom. Hertz[26] and also Lüdin[36] have reported that a barium meal will pass along the gut faster if it is followed by a second meal given a short time after the first. Observers have stated, furthermore, that contrast meals leave the stomach more rapidly if they are made palatable. One other instance was given me by one of my students, Mr. S. M. Wheeler. He had had an obscure inflammation of the intestines; during his convalescence he noted that whenever he took the first food of the day he had a griping sensation localized in the colon—if he had no breakfast the sensation did not appear until he ate lunch. Recently my colleague, Dr. Sosman, has told me that he has been able to stimulate gastric peristalsis quite markedly in the course of a fluoroscopic examination by talking to the patients in a tempting way about food which they like. Women, he reports, respond best to mention of salads and desserts, whereas men are more affected by remarks concerning such substantial viands as roast beef or ham and eggs or ice cream. From all this evidence the conclusion seems justified that the experience of eating, when it is enjoyed, or even the mention of pleasure-yielding food to which the individual has been conditioned, has a highly favorable effect on the functioning of the alimentary canal.

We must not suppose that any extrinsic nerves act specifically to produce peristaltic waves. The moving rings of constriction, as they course along the stomach and intestines, are displays of an inherent capacity of the neuromusculature of the part. But, as already indicated, this capacity depends on the state of "tonus," or, preferably expressed, the state of moderate tension of the musculature, and that normally is dependent on the extrinsic nerves. The evidence for that statement is demonstrated best in the esophagus. When the vagi are cut the whole gullet is paralyzed. Food accumulates in it until it has the appearance of a fusiform sac. As days pass, however, the lower part of the tube, which is composed of smooth muscle, gradually contracts more and more, establishing thus an intrinsic tonus, and as it does so it manifests more and more prominently and effectively peristaltic contractions. A similar transformation is observable in the stomach; the flabby, atonic condition which follows soon after vagotomy is associated with absence of peristalsis, and the reappearance of peristaltic waves is concomitant with the development of an intrinsic tone in the gastric wall.

I have laid considerable emphasis on the phenomenon of tonus, for two reasons. First, I wished to make clear the importance of paying attention to the satisfaction of appetite, to the agreeableness of the ingested food, for a proper starting of the digestive process, not only in relation to the secretion of the several glands, but also in relation to the muscular functions of the alimentary canal. Fluid which has no appetizing qualities—a barium "meal," for example—will yield pictures of peristalsis, no doubt, but there is good reason to question whether it should be used for judging what would happen in all cases,—as when, for example, really delicious food is taken instead, food that would make the mouth water! The second reason for the emphasis on tonus is that it relates the functioning of the digestive canal to the general condition of the body. Since I regard that relation as fundamentally important for bodily welfare, I wish, as a final consideration, to discuss it.

The Bearings of the General Bodily Condition on the Digestive Process. The stomach and intestines are not isolated portions of the body, I need hardly say, but are highly important members of the collaborating groups of organs which constitute the or-

ganism. They serve, and are served by, the other groups. Thus relations exist such that not only do the digestive organs suffer when the body suffers, but the body suffers in turn when they are disordered.

The most influential agency involving the alimentary canal with conditions in the organism as a whole is that portion of the nervous system which is concerned with internal affairs—the autonomic system. It is unnecessary to go into detail regarding the arrangement of this system; its distribution to smooth muscles and glands, its separation into three divisions (cranial and sacral or parasympathetic divisions, and the middle or sympathetic division), and the usual opposition of sympathetic to parasympathetic influences when they meet in any structure, are too well known to you.

Already I have mentioned the main representative of the cranial division that is of interest—the vagal supply, which extends from the esophagus over the stomach and throughout the small intestine. An analogous innervation of the colon is found in the pelvic visceral nerves of the sacral division.

Evidence, which we have already surveyed, that when food is relished the smooth muscle of the alimentary tract receives impulses by way of the vagi that excite it to activity, raises the question as to what may happen if these impulses are not delivered. The extreme condition, which is experimentally realized when the vagi are cut, indicates that muscular tone is lost. Is this loss of tone indicative of what occurs when the nerves are whole but are not properly functioning?

Observations which I reported in 1911[12] (p. 211) bear on this question. With the vagal connections between the alimentary canal and the nervous system intact, the general asthenia resulting from infection, and characterized by soft, toneless skeletal muscles, is associated with a toneless state of the muscular wall of the stomach and intestines. The animals manifest no signs of appetite; they do not eat spontaneously and must be forcibly fed. Animals in that condition were examined repeatedly with roentgen rays, and throughout a whole day the food was seen lying in the stomach, quite undisturbed by peristalsis; and any chyme which may have been pressed into the intestine was similarly unaffected because of inactivity of the gut. From what we have already noted regarding vagal influence on the gastrointestinal muscles we may assume that the indolence or total lack of movement of the muscular wall is due to absence of the tonus necessary for rhythmic contractions.

The observations made on lower animals have been confirmed in human cases. According to Alvarez,[2] autopsies on patients who have died from botulism have disclosed food in the stomach which had been eaten many days before, when the symptoms first appeared. Similar stagnation has been reported as present in the stomachs of persons suffering from tuberculosis and other infectious disease. Also exhausting labor is associated with failure of proper action of the digestive organs. In a man with a gastric fistula Mantelli[10] noted that for an hour or two after strenuous muscular exertion the stomach did not respond normally to the actual presence of food. These cases indicate that if there is a debilitated or asthenic state, as a consequence of exhaustion, whether from overwork or prolonged disease or acute infection, the gastrointestinal tract shares in the tonelessness of the rest of the body. In consequence, the digestive processes do not proceed vigorously. Thus a vicious circle may be started so that the debilitated organism does not receive the nutriment necessary for its upbuilding, and digestion is impaired because of the debilitation.

Digestion, besides being disturbed because in general bodily atony the cranial division of the autonomic system fails to establish a tonic condition of the alimentary tract, may be disturbed also because of the positive action of the sympathetic or middle division of the system. This division, having, as a rule, an effect opposed to that of the cranial, produces in the main

a lessening or inhibition of tone in the smooth muscle of the stomach and intestines. As is well known, the sympathetic is brought into special activity by various conditions, but commonly, by emotional excitement. The first trouble which I had, when studying the movements of the stomach in 1897, was due to an interruption of the peristaltic waves, or their failure to appear, because of this condition. Often the only other sign of excitement which the cat manifested was a restless turning of the body or a to-and-fro twitching of the tail. A gentle petting or a stroking of the fur would restore a state of calm, whereupon the waves would start again. At the time I thought it unfortunate that so beneficent a process as digestion should be so readily subject to interference. It seemed to me that the rational course to follow would be to excise the part of the sympathetic which distributes to the alimentary canal, in order that the digestive process might be freed from any check. Only after many years of investigation of that division of the autonomic system, during which were revealed its functions as a mobilizer of the bodily forces in various critical states[14] and as a regulator of the constancy of the blood and lymph,[15] did I see fully that the checking effect of sympathetic impulses on the stomach and intestines is only a small part of the total picture. Besides this effect on the alimentary canal sympathetic impulses cause a secretion of adrenine from the adrenal medulla, and then, in cooperation with adrenine in sympatho-adrenal service, they liberate sugar from the liver into the blood stream, shift the blood from the splanchnic area into dilated blood vessels of skeletal muscles, and raise blood pressure so that the flow is faster through the heart and brain and regions of activity; they dilate the bronchioles and make vigorous breathing easier; they contract the spleen and from its stores cause a discharge of extra red corpuscles into the blood stream; they accelerate the speed of blood clotting; they increase metabolism. All these and other remarkable changes attendant on emotional excitement are similar to the changes which occur when the body enters into strenuous exertion. They may properly be regarded as preparations for struggle, perhaps supreme struggle.[14] And further analysis indicates that the value of these alterations in the organism lies in the preservation of a steady state in the internal environment or fluid matrix of the body in spite of heat- and acid-production by contracting muscles and the using up of energy reserves during the struggle. If the sympathetic system is removed, the possibility of adapting the organism to demands from external and internal shifts of conditions is largely lost.[15] It appears, therefore, that the way of avoidance of digestive difficulties from worry and anxiety is not through the application of surgical skill but through the exercise of forethought and self-direction.

Emotional perturbation is only one of several conditions in which sympathetic impulses are aroused. Pain will arouse them; and also asphyxia and external cold and hemorrhage and low blood pressure. Long ago—in 1907—Murphy and I[17] made a report on the first stages of a research on the effect of "painful" stimuli in producing ileus—a research which was unfortunately interrupted and not later resumed. We found that when ileus was produced thus reflexly physostigmine salicylate had a temporarily beneficial influence in exciting peristalsis—an influence which Ritvo and Weiss[44] have emphasized. This initial study on ileus should be taken up where we left it and carried to completion. And there are chances also for studies on the influence, on the movements of the alimentary tract, of external cold, of asphyxia, and of hemorrhage and low blood pressure (both of which may be regarded as involving tissue asphyxia). My colleagues, Freeman, Morison and Sawyer[22] have shown that loss of blood volume by dehydration—a state closely related to surgical shock—is attended by increased functioning of the sympathetic system. Is the alimentary canal involved in this reaction? Does post-

operative ileus result in part from dehydration during the operation? These are interesting and important questions which call for answers.

My chief reason, however, for stressing at this time emotional states rather than these other circumstances as stimulators of sympathetic impulses, and therefore as disturbers of gastrointestinal functions, is that in some individuals the emotional reaction is extraordinarily sensitive. Todd[47] has recorded instances of sudden lessening of the tone of the stomach, and inhibition of peristalsis, not only from fear but also from slight alarm because of sudden and unusual occurrences during a roentgenographic exposure. Such conditions are likely to be misinterpreted. Obviously they are of prime significance in the understanding of digestive disorders and in a rational interpretation and treatment. It would be well if we knew more of the circumstances of their appearance, the types of patients in whom they are most likely to appear, and the best modes of assuring their avoidance or their cure.

In this survey of researches carried on many years ago I have availed myself of the privilege accorded a person who has passed into the seventh decade of life—the privilege of engaging in reminiscences. It has been a real pleasure to me to return for a time to former interests and to discuss them with you. In doing so I have endeavored to indicate where further research might prove fruitful. Perhaps some among you may be tempted by these hints to undertake studies which would yield the information we still need. That would be, I am sure, wholly in accord with the spirit of Dr. Caldwell.

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A ROENTGENOLOGIC STUDY OF TUBERCULOSIS OF THE LARYNX AND NECK*

By HENRY K. TAYLOR, M.D., F.A.C.P., *and* LOUIS NATHANSON,† M.D.

NEW YORK CITY

LARYNGEAL tuberculosis is a frequent concomitant of the malignant type of pulmonary tuberculosis.** It occurs often enough to warrant routine roentgen studies of the neck, in view of their diagnostic value.

HISTORICAL

Thost in 1913 and again in 1932 claimed to be able to detect early tuberculous lesions and asserted that a differential diagnosis between tuberculosis and syphilis could be made on the roentgenologic findings. He attached great value to the roentgen examination because it makes possible the determination of early lesions within the ventricle which might be overlooked on mirror examination.

In 1914 Iglauer, referring to laryngeal tuberculosis, commented on a "washed-out" appearance of the cartilages and infiltration of the soft tissues, an observation in which Thost concurred. Dr. Sidney Lange, who made the roentgen studies for Iglauer, described his technique and the normal larynx.

Pancoast specifically states that an obliteration of the laryngeal ventricle may be due to a new growth, an inflammatory process or paralysis of a vocal cord. In his opinion there is no roentgenologic distinction between tuberculosis, syphilis and tumors. Pendergrass shares this view.

Chevalier L. Jackson observed that inflammatory and neoplastic lesions can be recognized early, with only the mildest symptoms present. He presented 2 cases in which the roentgen examination showed abnormal findings in the larynx, without diagnosing any specific lesion.

Hay, in his excellent monograph, lists 6 cases of tuberculosis of the neck. Four of these were glandular and the other 2 ulcerative lesions.

In a comprehensive article on the value of roentgenologic studies of the neck, Sussman mentions many laryngeal and extralaryngeal lesions. Wotzilka calls attention to the value of roentgen examination of the larynx and stresses the importance of the appearance of the cartilages.

Ziegelman considers tuberculosis the most common specific lesion affecting the larynx. In his opinion laryngeal tuberculosis rarely presents itself as tuberculoma.

Patton in 1930 pointed out that laryngeal tuberculosis has its greatest incidence between the ages of twenty and forty, although it is not rare after the latter year. He remarked that subjective symptoms may be entirely absent in slight or early cases.

PATHOLOGY

The cervical lymph nodes, especially the submaxillary glands, are the most frequently affected by tuberculosis. They are, as a rule, secondarily involved from foci in the tonsils or upper respiratory passages. Single nodes may be involved, but most frequently they coalesce and become matted together forming a swelling on the side of the neck. At times chains of glands are found.

These glands show a tendency to softening and caseation and they frequently burrow through the skin and establish a discharging sinus. The necrotic material, instead of softening, may become dry, hard and undergo calcification.

Tuberculous lesions of the spine are

** Caseous-pneumonic type of tuberculosis in contradistinction to (a) the benign exudative, (b) the exudative-productive and (c) the chronic proliferative types. (Classification of Ornstein, Ullmer and Dittler.)

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† Thesis submitted to the Faculty of Radiology of the Graduate School of Medicine of the University of Pennsylvania (1934) in partial fulfillment of the requirements for the degree of Master of Medical Science (M.Sc.(Med.)) for graduate work in Radiology.

usually secondary to a tuberculous focus elsewhere. The route of infection may be hematogenous from a distant focus or lymphogenous from a contiguous glandular involvement. The body of the vertebra is nearly always the site of involvement, the appendages being rarely involved. Three types of involvement of the body are recognized: (1) The central type which, according to Steindler, constitutes the greatest proportion of cases in children. Frequently this lesion causes complete collapse of the body of the vertebra. (2) The epiphyseal type where the upper or lower borders of the vertebra in the region of the epiphyses become involved. In this type there is an early involvement of the intervertebral cartilages with absorption of the disc and obliteration of the joint space. (3) Anterior peripheral type. Here the anterior surface of the vertebra becomes involved and the lesion subsequently extends into the body.

Cold abscess formation is a complication of Pott's disease. It manifests itself in the form of a dense paravertebral swelling. In the cervical region the abscess frequently appears in the prevertebral soft tissues, which thicken markedly, depending on the size and extent of the abscess. This abscess encroaches upon the air spaces and at times displaces the larynx and trachea anteriorly. A swelling of the prevertebral soft tissues may be present without abscess formation, and is a contiguous infection from the spine or larynx.

The pathology of laryngeal tuberculosis manifests itself grossly as infiltration, edema, ulceration, tumor formation and perichondritis. The laryngeal structures involved in the order of their frequency are the interarytenoid space, arytenoids, vocal cords, ventricle and ventricular space, aryepiglottic folds and epiglottis.

According to Wood, infiltration is the earliest stage of laryngeal tuberculosis, and may involve any part of the larynx. Edema is frequently associated with laryngeal tuberculosis. It is seldom found involving the true or false cords, due to the absence of loose areolar tissue. It most frequently involves the arytenoid eminences, aryepiglottic folds and epiglottis.

Kaufmann describes three types of ulceration: (*a*) superficial lenticular ulcers, (*b*) deep ulceration which follows disintegration of tuberculous infiltrates in the depths of the mucosa and (*c*) ulcers running a chronic course which show a tendency to become perichondrial.

Perichondritis develops with direct extension of the lesion into cartilage. The perichondrium separates from the underlying cartilage and an abscess may form, or an adhesive perichondritis develops which results in a thickening of the cartilage (Turner). The arytenoid cartilage is the one most frequently involved.

Masses of tuberculous granulation tissue may form small tumors or tuberculomas. They are generally found in conjunction with tuberculous ulceration in other parts of the larynx. They have been found attached to the posterior portions of the cords, interarytenoid area and limiting mucous membrane of the ventricle and vestibule. Two types are recognized: (1) fibrotuberculoma, where connective tissue elements predominate, and (2) granulotuberculoma, in which the tubercles are surrounded by granulation tissue.

In the interarytenoid region one first notes a thickening and heaping up of mucous membrane which may assume the appearance of a tumor formation. It is nearly always papillomatous in character. It may eventually become ulcerated. The area in front of the arytenoid cartilages is a common site for tubercle formation.*

The arytenoid eminences (Wood, Turner) become swollen and edematous due to the involvement of the lax submucous tissue. The swelling may become extensive and overhang the glottis. Ulceration, while it may occur, does not frequently involve the arytenoid eminences.†

* Stoerk (quoted by Turner) first pointed out that this interarytenoid involvement was almost pathognomonic of tuberculosis when found in conjunction with pulmonary tuberculosis.

† Wood in describing the lesion states that this appearance is characteristic of tuberculosis.

Infiltration of the cords rarely occurs. When it does, it is usually found in the middle or posterior portion. At times minute ulcerations occur along the edges of the cords giving them a "mouse-eaten" appearance. These ulcerations may go on to destroy considerable tissue and they are most common over the posterior half of the cord. Vegetations sometimes arise from the ulcer and may encroach upon the glottis and at times overhang into the subglottic area.

The ventricular bands are subject to the same pathological changes as the true cords. Small lesions may develop in the ventricle, gradually increase in size and project into the glottis.

Where the aryepiglottic folds are involved, the infiltration may be uniform. Often it is most marked at the arytenoid end. The folds may become thickened to many times their normal size and both surfaces may be involved. When ulceration occurs it is most frequently found on the pharyngeal surface and at the arytenoid end.

The epiglottis, when involved, may show a thickened free border or a generalized swelling to several times its normal size. It appears as a thick, rigid, dense mass. Ulceration occurs either along the free margin or in the main mass of the epiglottis. Ulceration results in loss of tissue and the extent varies with the degree of involvement.

Lesions in the subglottic region are usually extensions from above and may be overhanging granulation tissue from the cords or ventricle. Sharply circumscribed local infiltrations are found on the under surface of the cords and at the upper portion of the trachea.

TECHNIQUE

The patient sits erect in a straight-back chair with his head supported in a head rest. The shoulders are dropped and drawn back with one shoulder resting against the cassette. The mandible is held horizontal to the floor and the chin is extended forward. The head and body are placed in true lateral position. As a rule an 8″×10″ film is used in double intensifying screens. The central ray is projected through the midportion of the thyroid cartilage. The following factors are used; 55 kv., 100 ma., at 6 foot distance, with an exposure time of 1/20 or 2/20 of a second, depending on the thickness of the neck. When the thyroid cartilages show considerable calcification, oblique positions are taken.

All cases presenting themselves for roentgen studies of the neck have a complete roentgenoscopic examination, which includes observations during phonation, inspiration, expiration, and swallowing, with and without barium. This is necessary to determine the mobility of the arytenoid eminences as well as the larynx as a whole. We wish to caution, at this time, that complete accommodation is absolutely necessary before starting the roentgenoscopic examination. The routine roentgenographic examination follows and this consists of two lateral views taken by teleroentgenography. One exposure is made with the larynx at rest and the other during phonation, employing a high-pitched E sound. From our experience this is the minimum requirement for the detection of pathological changes. When pathology is found complete detailed roentgenographic examinations are made in addition to the roentgenoscopic study.

A roentgen study of the neck in the lateral view yields considerable useful information. At the present time this may not always lend itself to the absolute differentiation of the hyperplastic and ulcerative lesions (inflammatory and neoplastic), but with further study greater accuracy may be achieved. Study of the soft tissues and air spaces of the neck yields data which can be corroborated by the laryngologist. It supplements the available clinical data and is a valuable record which should be made prior to the clinical examination.

The structures visualized on a lateral roentgenogram of the neck are divided into four groups:

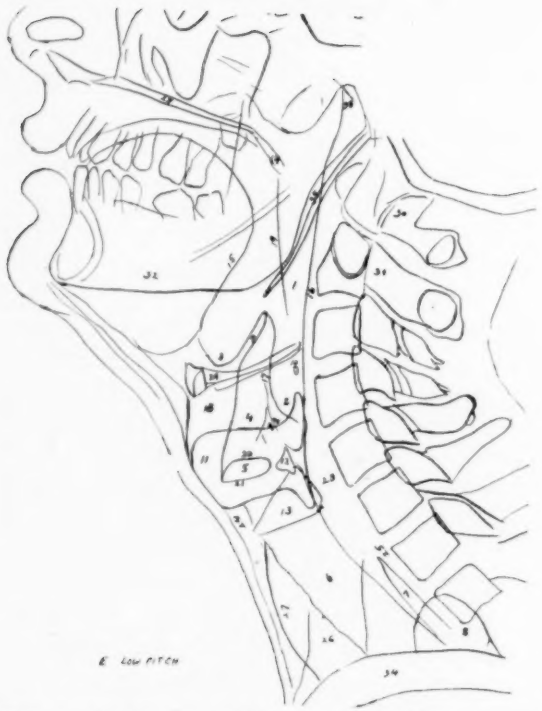

Fig. 1. *Air Spaces:* 1. Pharynx. 2. Pyriform sinuses. 3. Epiglottic vallecula. 4. Laryngeal vestibule. 5. Laryngeal ventricle. 6. Trachea. 7. Air in cervical esophagus. 8. Apex of lung. *Cartilages:* 9. Epiglottis. 10. Cartilago triticea. 11. Thyroid cartilage. 12. Arytenoid cartilage. 13. Cricoid cartilage. *Soft Tissues:* 14. Uvula. 15. Tongue. 16. Posterior pharyngeal wall. 17. Aryepiglottic fold. 18. Fat pad between epiglottis and thyrohyoid membrane. 19. Pharyngopalatine fold. 20. False cord. 21. True cord. 22. Arytenoid eminence. 23. Postcricoid area. 24. Cricothyroid membrane. 25. Retro-tracheo-esophageal area. 26. Sternomastoid muscle. 27. Thyroid gland. *Bones:* 28. Hard palate. 29. Hyoid bone. 30. Atlas. 31. Axis. 32. Mandible. 33. Mandibular condyle. 34. Clavicle. 35. Styloid process.

I. *Bones*
- A. Cervical spine
- B. Hyoid bone
- C. Mandible, styloid processes, base of skull, clavicle, etc.

II. *Cartilages*
- A. Epiglottis
- B. Thyroid
- C. Cricoid
- D. Arytenoids

} when calcified

and

Small adjacent cartilages (Santorini, cuneiform, Wrisberg) at times
- E. Cartilago triticea at times
- F. Tracheal rings when calcified

III. *Soft tissues*
- A. Arytenoid eminences
- B. Glands
  - Submaxillary, Thyroid, Lymph nodes } When enlarged or calcified
- C. Muscles
- D. Prevertebral tissues
  - Posterior pharyngeal wall
  - Postcricoid area
  - Retro-tracheo-esophageal area

IV. *Air spaces*
- A. Pharynx
  - Nasopharynx
  - Oropharynx
  - Epiglottic vallecula
  - Pyriform sinuses
- B. Larynx
  - Vestibule
  - Ventricle
- C. Trachea
- D. Apex of lung
- E. Cervical esophagus

The air in the neck is the equivalent of a contrast substance and enables one to visualize many structures in the lateral roentgenogram. In a general way, these air spaces are subdivided into three main parts: the pharynx, the vestibule of the larynx, ventricle and the subglottic region.

The pharynx appears as an irregular space bounded anteriorly and superiorly by the posterior surface of the tongue, the uvula and the base of the skull. The epiglottic vallecula, the epiglottis and the aryepiglottic folds form its lower anterior boundary. Its posterior boundary consists of the soft tissues forming the posterior pharyngeal wall, which extends from the base of the skull to the pyriform sinuses.

The vestibule of the larynx is triangular in shape. It is bounded anteriorly by the epiglottis and posteriorly by the aryepiglottic folds above and the arytenoid eminences below. The apex of the triangle is formed by the junction of the epiglottis and the aryepiglottic folds. The ventricular bands of the larynx form its base.

The laryngeal ventricle is an oval aerated space, with more or less convex upper and lower borders, situated between the true and false cords. The ventricular space

extends from the thyroid cartilage anteriorly to the vocal processes of the arytenoid cartilages posteriorly.

The cords are visualized as more or less sharply defined bands which move during phonation, changing the size and shape of the ventricular cavity. The upper margins of the false cords and lower margins of the true cords are not sharply defined but gradually taper off into the adjacent structures.

The subglottic region extends downward from the lower margin of the ventricle and includes the cervical trachea, the apex of the lung and the cervical esophagus. Air in the cervical esophagus and apex of the lung is not always visualized.

Hay, in his excellent monograph on "The Neck," gives detailed measurements of the various structures, which we have corroborated. There is practically no change noted when using teleroentgenography as compared to the 48 inch distance used by Hay.

The size and shape of the air spaces in the neck are not constant. They show marked changes during phonation, change in pitch, coughing, swallowing, whistling and on movement of the head. With a high-pitched sound the larynx becomes elevated and there is a corresponding increase in the length of the cervical trachea.

Fig. 2*a*. C.B., male, aged twenty-three. Normal larynx roentgenographed during phonation—low-pitched E.

Fig. 2*b*. Normal larynx (same as Fig. 2*a*) roentgenographed during phonation—high-pitched E. Compare with Figure 2*a* and note elevation of larynx and change in contour of the air spaces.

Roentgenographic pathology in tuberculous lesions of the neck:

I. *Glands*
  (a) Calcified lymph nodes
  (b) Non-calcified lymph nodes

II. *Bone*
  (a) Spine—caries

III. *Prevertebral soft tissues*
  (a) Postpharyngeal wall
  (b) Postcricoid area
  (c) Retro-tracheo-esophageal area
  (d) Cervical esophagus

(a), (b), (c) — Thickened or cold abscess

IV. *Larynx*

(a) Minimal
   1. Ventricular
   2. Interarytenoid

(b) Moderate
   1. Arytenoid area, with or without involvement of adjacent tissues

(c) Extensive
   1. All laryngeal structures with or without involvement of adjacent tissues

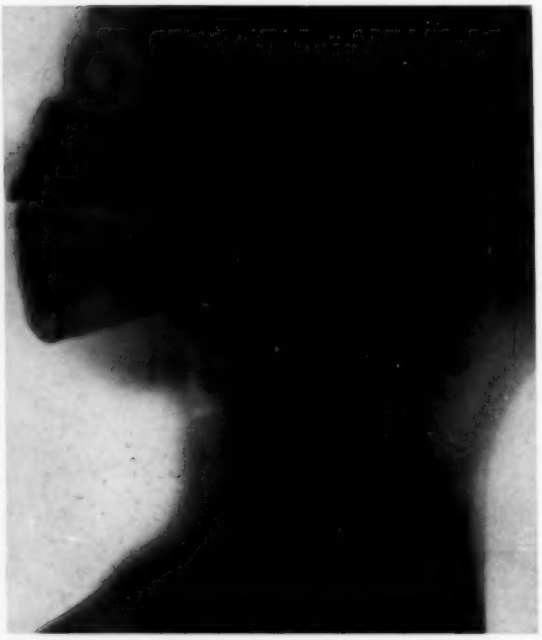

Fig. 3. T.D., male, aged four. Retropharyngeal cold abscess.

Tuberculous lymph nodes are usually matted together and form large masses. At times single nodes are visualized. Calcareous deposits in nodes are visualized and are of interest when many are involved. We have seen chains of calcareous deposits on either side of the neck, involving all the cervical and supraclavicular as well as the axillary lymph nodes. Calcareous deposits in nodes are usually considered as due to tuberculosis, and excludes other causes for adenopathy.

The cervical spine requires no comment and is included for completeness. Caries of the cervical spine does occur and may be present with or without a cold abscess.

The posterior pharyngeal wall in adults is normally thin and measures about 2 to 3 mm., or about one-fifth the width of the body of the fifth cervical vertebra (Hay). At times, accompanying laryngeal tuberculosis, there is a slight thickening of the posterior pharyngeal wall and it exceeds its normal measurement. When the posterior pharyngeal wall is unusually thickened, to many times its normal measurement, a retropharyngeal abscess is to be considered. A new growth or glandular enlargement must be excluded. Sometimes a retropharyngeal abscess is huge and bulges, almost completely obliterating the entire pharynx and exerting pressure upon the laryngeal structures. Such abscesses may persist for months. A retropharyngeal cold abscess may extend into the thorax. When it does there is marked thickening of the retro-tracheo-esophageal soft tissues. These cold abscesses may be present alone or with a tuberculous lesion involving the larynx and cervical spine, either separately or jointly. In several instances autopsy revealed caries of the spine, not demonstrable on roentgen examination.

We have had one proved case of tuberculosis of the esophagus and no characteristic roentgen changes were found. This case will be reported by Dr. Mervin C. Myerson in detail in the near future.

The laryngeal tuberculous lesions are quite common and occur in a high percentage of patients with the malignant type of pulmonary tuberculosis. No portion of the larnyx is immune from involvement. The lesion manifests itself as a swelling or thickening of one or more of the various laryngeal structures which may be due to edema or infiltration. Localized swellings or tuberculomas may occur. Destruction of any of the structures is due to ulceration. The lesion may extend into any of the laryngeal cartilages. At the present time we classify the lesion into three groups depending upon their extent rather than on an anatomical or pathological basis. In most instances, however, these subdivisions, namely, minimal, moderate and

extensive, embrace given anatomical structures. The minimal lesions in our grouping, for example, are located anatomically either in the ventricle or in the interarytenoid area or both. The group of moderate lesions involves the arytenoid eminences with extension either above or below them, i.e., into the interarytenoid area below and the aryepiglottic folds above. Adjacent soft tissue may also be involved. The group of extensive lesions affects practically all the laryngeal structures, with or without involvement of the contiguous tissues. Involvement of the epiglottis is particularly pronounced in this group.

In the minimal group, a small localized swelling or tuberculoma may be seen in the ventricle as a small circular or oval dense shadow which encroaches upon the ventricular air space. At times it is attached to the true cord and projects into the ventricle. It may be attached deep in the ventricular pouch or on the ventricular band. The tuberculomas may project into the laryngeal vestibule or into the trachea. They may obliterate the anterior or posterior commissure, according to its location. Ventricular obliteration depends upon the size of the tuberculoma. In our experience this is an infrequent lesion. Our findings in this respect coincide with those of Ziegelman, who claims that tuberculomas are not common.

Most of the minimal lesions are in the interarytenoid area, which is not visualized on the roentgenogram. This is the space between the arytenoid cartilages and the arytenoid eminences, posterior to the ventricles. It may be referred to as the dark space. With the larynx at rest most of the minimal lesions in the interarytenoid area cannot be detected. During phonation, using a high-pitched E sound, there is shortening of the interarytenoid area, an elongation and narrowing of the ventricle and an elevation of the larynx. If a minimal lesion is present there may then be observed a distortion of the posterior portion of the ventricle, whose contour becomes indistinct in contrast to the sharp

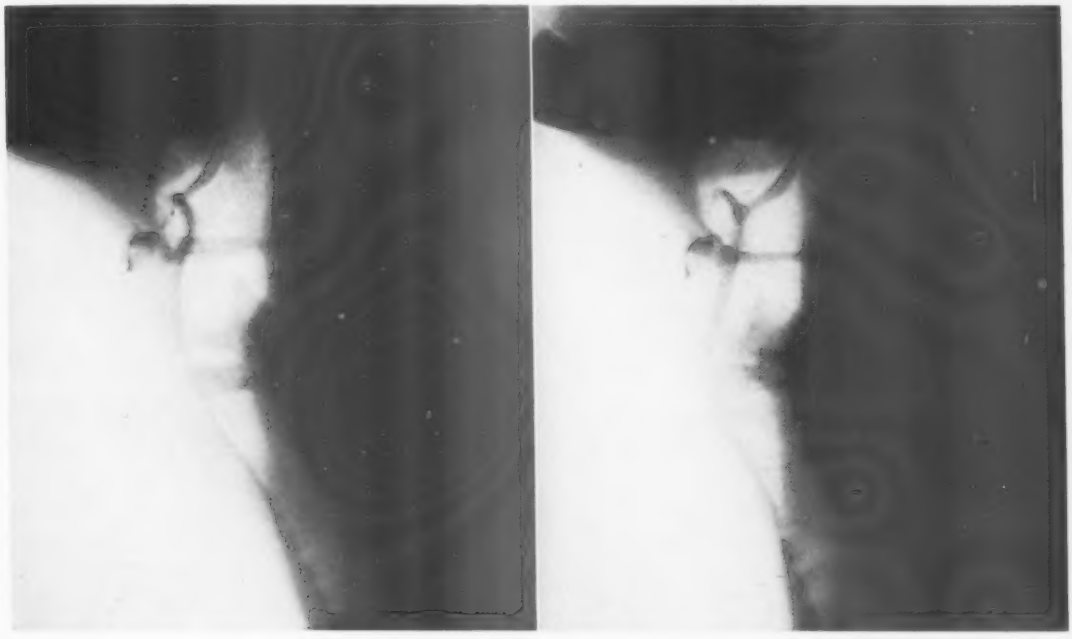

Fig. 4*a*. A.H., female, aged thirty. Larynx at rest. Ventricle appears normal. Non-calcified gland anterior to fifth cervical vertebra, overlying posterior border of trachea.

Fig. 4*b*. Roentgenographed during phonation—E. Miminal lesion. Note ventricular change. Clinical examination: Small lesion in interarytenoid area.

Fig. 5*a*. S.F., male, aged thirty-one. Larynx at rest.

Fig. 5*b*. Roentgenographed during phonation—E. Miminal lesion. Compare with Figure 5*a* and note ventricular changes. Clinical examination: Cords thickened and injected. No ulceration.

Fig. 6*a*. S. Z., male, aged twelve. Larynx at rest. Aryepiglottic folds thickened. Slight irregularity in false cord. Posterior pharyngeal wall and postcricoid area thickened.

Fig. 6*b*. Roentgenographed during phonation—high-pitched E. Moderate lesion. Swelling of arytenoid eminences. Aryepiglottic folds thickened. Ventricle narrow and indistinct. The lesion appears more pronounced during phonation. Compare with Figure 6*a*. Clinical examination: Arytenoids reddened and infiltrated. Infiltration in interarytenoid area. Ventricular bands thickened. True cords thickened and red.

outlines of the anterior portion. The ventricle may be narrowed and slit-like. There is a loss in the aeration of its posterior portion, or the entire ventricle may become indistinct. As previously stated, there is no change in the appearance of the normal ventricle, other than in shape, when the larynx is studied at rest and during phonation. It is necessary to have a careful roentgenoscopic study to determine the free mobility of the arytenoid eminences as well as roentgenograms both at rest and during phonation for purposes of comparison when looking for these small minimal lesions. We have verified the above so often that we consider a single roentgenogram of the larynx of little value.

At times the lesion in the interarytenoid area may extend forward sufficiently to deform the contour of the ventricle. We have observed a deformity of its upper posterior boundary, which is normally convex throughout its entire extent.

In the moderate group the pathology noted is quite marked. Either one or both arytenoid eminences may be involved, usually both. There is sometimes considerable enlargement of these eminences, with smooth outlines due to edema. When they are enlarged and irregular, it is usually evidence of infiltration and ulceration. The aryepiglottic folds appear markedly thickened when edema or infiltration or both extends upward into them. A downward extension of the lesion causes ventricular changes. The ventricle may show various changes in outline: it may be narrowed down to a slit, become indistinct or be entirely obliterated. Here again, study of the ventricle at rest and during phonation is essential as either study may disclose ventricular changes not visible on the other.

Fig. 7. A.S., male, aged twenty-six. Roentgenogram taken during phonation—E. Moderate lesion. Arytenoid eminences markedly enlarged. Ventricle deformed by intraventricular localized tumefaction or tuberculoma. Postcricoid area thickened. Clinical examination: Arytenoids markedly swollen. Marked thickening of left false cord so that left true cord is not perceptible. Thickening of true and false cords, right side.

Fig. 8. F.M., male, aged twenty-seven. Larynx at rest. Extensive lesion. Marked swelling and thickening of tip of epiglottis, aryepiglottic folds, arytenoid eminences and laryngeal vestibule. Ventricle deformed by intra- and extra-ventricular lesion. Postcricoid area thickened. Clinical examination: Epiglottis swollen. Arytenoids congested and edematous. Small amount of exudate in arytenoid space. Fixation of crico-arytenoid joint. Opening of glottis small.

Ulceration of the cords shows irregular defects sometimes surrounded by localized swellings. The lesion may extend into the adjacent tissues, particularly the postcricoid area, which appears thickened on the roentgenogram.

A lesion in the ventricular pouch may show changes on the roentgenogram which appear to involve the true and false cords, particularly if the ventricle is narrowed. This is merely a superimposition of the ventricular lesion on the cords.

The extensive lesions show massive involvement of the laryngeal structures. The involvement, as a rule, appears regular, uniform and homogeneous, with a minimal amount of distortion. The epiglottis is usually thickened, rigid and erect and shows a marked increase in density. The aryepiglottic folds are also thickened and stand out as prominent bands. These changes are due to edema and infiltration. The involvement of the arytenoid eminences and ventricle is usually more marked than in the group described as moderate lesions. The ventricular space may not be completely obliterated, depending upon the extent of involvement of the true and false cords. In a small number of cases there is no ventricular involvement.

The epiglottis is shorter and has an irregular upper border when ulcerated.

FIG. 9. A.B., male, aged twenty-eight. Roentgenographed during phonation—E. Extensive lesion. Extensive swelling and thickening of all laryngeal structures. Tip of epiglottis thickened and slightly irregular. Ventricle is narrow, irregular, deformed and indistinct. Subglottic extension—upper posterior portion of trachea. Clinical examination: Epiglottis swollen and ulcerated. Arytenoids are swollen.

FIG. 10. H.P., male, aged thirty-eight. Larynx at rest. Extensive lesion. Extensive swelling and thickening of all laryngeal structures. Ventricle is deformed. There is marked thickening of the posterior pharyngeal wall, postcricoid and retrotracheo-esophageal areas. Subglottic extension—upper posterior portion of trachea. Cervical spine shows no evidence of caries. Clinical examination: Granular appearance of posterior pharyngeal wall. Epiglottis thickened and edematous making it possible to see only an edematous right arytenoid and no other laryngeal structures.

Involvement of the adjacent soft tissues produces a loss of laryngeal mobility. This is ascertained by roentgenoscopy or by comparing a roentgenogram of the larynx at rest with one taken during phonation.

A perichondritis or involvement of the thyroid cartilage cannot always be detected. If the cartilage normally shows

little or no calcification, the perichondritis cannot be recognized roentgenologically. Even if the cartilage shows considerable calcification, a perichondritis can only be suspected or detected by changes in the normal deposition of calcium. There may, for example, be an irregular area showing no calcification where one would normally expect to find it in considerable quantities. Calcification is a physiological process incidental to age.

Pancoast states that "infiltration of the cartilages cannot be determined unless there is extensive ossification which is, however, irregular and deceptive." Infiltration in the arytenoid eminences and epiglottis can be detected in the absence of calcification. In only one instance, in a man past seventy, did we find a calcareous deposit in the epiglottis, and this individual had no tuberculous lesion of the larynx or lungs. Pancoast states that one is never

FIG. 11. M.R., male, aged thirty-seven. Roentgenogram taken during phonation—E. Extensive lesion. Extensive swelling and thickening of epiglottis, aryepiglottic folds and arytenoid eminences. Irregularity of the tip of epiglottis (perichondritis). Ventricle irregular. Clinical examination: Epiglottis infiltrated and distorted. Remainder of larynx cannot be seen due to overlapping fixed epiglottis.

FIG. 12. I.J., female, aged twenty-six. Larynx at rest. Extensive lesion. Swelling and thickening of all laryngeal structures. Irregularity of tip of epiglottis with loss of tissue (perichondritis). Ventricle distorted and almost completely obliterated. Subglottic extension of laryngeal lesion. Posterior pharyngeal wall and postcricoid area thickened. Cervical spine shows no evidence of caries. Clinical examination: Epiglottis is markedly infiltrated, about one-fourth of free edge is sloughed away. Arytenoids are infiltrated and ulcerated. True cords cannot be visualized due to fixation of epiglottis.

The decalcification or washed-out appearance of the laryngeal cartilages reported in the literature was not observed in our cases. The density of the thyroid and cricoid cartilages is of little value in diagnosing laryngeal tuberculosis, irrespective of the amount of calcification, unless a perichondritis is suspected.

justified in reporting cartilaginous erosions unless there is evidence to indicate involvement of the adjacent soft tissues. However, the reverse is not always true, soft tissue involvement does not mean perichondritis. We have observed prevertebral soft tissue involvement without clinical or roentgen evidences of perichondritis.

We have seen subglottic extensions only in association with lesions of the glottis. They appeared as localized swellings or masses in the lumen of the trachea, along its upper posterior wall immediately below the glottis. Eight cases presented subglottic lesions; 3 proved to be sharply defined tuberculomas and 5 were extensive edemas.

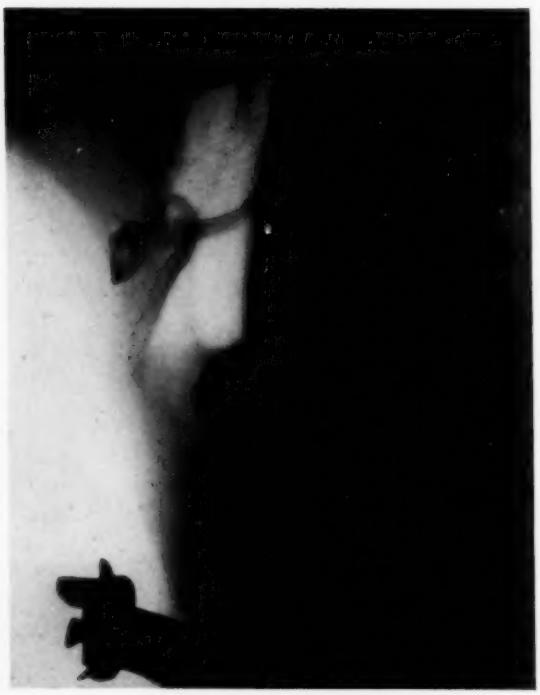

FIG. 13. Z.C., female, aged eighteen. Larynx at rest. Moderate lesion—laryngeal stenosis. Ventricle is obliterated. Marked narrowing of laryngeal vestibule due to swelling of arytenoid eminences posteriorly, and thickening anteriorly. Subglottic region narrowed by extension of the lesion. Lower portion of epiglottis appears ulcerated. There is no involvement of the tip of epiglottis or aryepiglottic folds. Tracheotomy tube in situ. Clinical examination: Marked edematous swelling of left arytenoid which projects beyond the midline. The right arytenoid is thickened. Ulceration of left vocal process. False cords thickened and ulcerated. Only the anterior portion of left true cord visible.

In 2 cases we have observed laryngeal stenosis. Neither of these showed extensive involvement but both presented lesions in the ventricular region which resulted in stenosis of the glottis.

There is a slight discrepancy between the clinical and roentgen findings in minimal lesions. A few of the latter are missed on roentgen study, when the lesions are small and are situated deep in the interarytenoid area. The lesion must reach a given size in this location before we can detect pathology, or else it must be situated more anteriorly, nearer the ventricle.

On the other hand, in our series roentgen examination discovered one case which had been missed clinically. Here the lesion was deep in the ventricular pouch. Wood claims that ventricular lesions are often missed by the mirror examination. Quoting him: "The most difficult lesion to diagnose is the early infiltrative infection of the ventricle of Morgagni." Because of this he stresses the value of roentgen study. Comparing the latter with the clinical examination in minimal lesions, small ventricular lesions can be missed clinically, while small interarytenoid lesions can be missed on roentgen study.

In the moderate and the extensive lesions, there is very little difference between the clinical and the roentgen findings in the larynx, although we may differ slightly as to pathological details. Small ulcerations may be missed on the roentgenogram for they are overshadowed by the adjacent pathological changes. We cannot detect redness or injection or congestion of the epiglottis or arytenoids. However, because of the sagittal projection, we are able to recognize height, width and subglottic extensions, as well as extralaryngeal involvements, while the clinician sees only the surface. He may not see the laryngeal vestibule or cords if there is marked edema of the epiglottis; he cannot see any subglottic extension and he necessarily misses extralaryngeal lesions unless they bulge into the pharynx. We found 15 cases with thickening of the prevertebral soft tissues which had not been perceived on mirror examination.

Laryngeal lesions are very often missed unless one looks for them. Routine roentgen examination of the neck revealed 8 cases with laryngeal tuberculosis which

were symptom-free; 3 with minimal, 4 with moderately extensive and 1 with extensive involvement of the larynx. In none of these cases was there involvement of the cords or ventricle.

On the negative side again, the roentgenologist cannot always tell detailed pathology as it affects the cords, although they may appear irregular or indistinct. Small ulcerations, for instance, may be obscured by the superimposition of adjacent pathological changes.

We have differed at times with laryngologists as to the presence or absence of edema of the arytenoid eminences. From the roentgenological standpoint, the arytenoid eminences show evidence of edema when they appear enlarged and the upper surfaces are smooth, round, regular and convex.

All in all, the roentgen examination gives a permanent pictorial record of the location and extent of the lesion. Serial or interval studies permit comparison and the visualization of changes in the pathological process.

According to Wood, Turner, Thompson and others, in the large majority of cases, unless the lesion seen in the larynx is very characteristic of some other condition, the presence of pulmonary tuberculosis is very good evidence that a suspicious involvement of the larynx is tuberculous. In our series of cases roentgen and clinical studies of the chest were positive for tuberculosis in every instance except one. In this case the laryngeal lesion was secondary to a Pott's disease of the upper dorsal spine. On the other hand, the presence of a pulmonary lesion does not rule out the possibility of either syphilis or cancer being present and it is possible to have mixed lesions.

The clinical side of all our laryngeal cases was taken care of by Dr. Mervin C. Myerson and his staff. Biopsies were performed in all cases where consent was obtained, provided there were no contraindications. Biopsies were obtained in 20 cases and 9 cases in which biopsies were unobtainable subsequently came to autopsy.

During the past two years we found on routine examination 5 cases of malignancy of the larynx, 3 papillomata, 4 cysts of the larynx and 1 case of syphilis. This later developed laryngeal stenosis. In all the above cases, except in 2 instances, there was positive roentgen and clinical evidence of pulmonary tuberculosis. Two cases presented laryngeal cysts and laryngeal tuberculosis.

DIFFERENTIAL DIAGNOSIS

The roentgen examination of the neck may not always lend itself to the absolute differentiation between laryngeal tuberculosis, syphilis, benign and malignant neoplasms, paralysis of the vocal cords, etc. Authorities generally agree, however, that in the large majority of cases, unless the lesion seen in the larynx is very characteristic of some other condition, the presence of pulmonary tuberculosis is very good evidence that the pathological involvement of the larynx is tuberculous. In many instances, the site of involvement and gross pathological appearance visualized on the roentgenogram suggests the diagnosis.

Syphilis as a rule attacks the front of the larynx, whereas tuberculosis in the early stages attacks the posterior portion of the larynx. The epiglottis is seldom attacked by tuberculosis until a late stage when other parts of the larynx are affected. The involvement is more marked on the pharyngeal surface. Syphilis shows a predilection for the anterior surface of the epiglottis and involves this structure early in the disease. The anterior portion of the cords are similarly a site of predilection. Kaufmann states that a frequent finding is the anteflexion of the epiglottis, due to scar tissue contraction and at times due to adhesions to the tongue. This is in contradistinction to the rigid, erect epiglottis frequently found in advanced laryngeal tuberculosis. Luetic lesions produce cicatrices and synechiae which deform the laryngeal structures and may completely obliterate the lumen. In tuberculosis, on

the other hand, there is frequently a minimal amount of distortion.

Iglauer, Thost, and others, state that syphilitic perichondritis is characterized by increased proliferation and calcification of the cartilages. Hay mentions one case of tertiary syphilitic perichondritis which showed this involvement, and comments that this finding may be true in the later stages of tertiary syphilis but not in the earlier lesions in which destructive changes predominate.

Benign tumors such as papillomata, fibroma, cysts and lipoma must be differentiated from tuberculoma. These tumors appear as sharply circumscribed and well-localized tumefactions arising from the anterior and middle portions of the vocal cords and ventricular bands and may encroach upon the air spaces of the vestibule of larynx, ventricle or subglottic region. As previously mentioned, tuberculomas are rare and though having a similar appearance one can frequently demonstrate changes in the surrounding tissue, where other structures appear thickened and swollen. They arise most frequently in the interarytenoid region and posterior portion of the cord as well as in the interventricular pouch. In our series, 4 cystic tumors were found, 3 of which arose from the epiglottis and 1 from the aryepiglottic fold.

Malignant infiltration may appear as a definite tumor, which frequently presents an irregular outline on the surface. Intrinsic malignant tumors most frequently arise from the anterior and middle portion of the cord. Extrinsic tumors nearly always arise from the pyriform sinuses, postcricoid area and from the posterior surface of the tongue and vallecula.

Malignant lesions may grow to be large massive tumors which deform and destroy en masse. When in the region of the ventricle they rapidly obliterate the ventricular space. It is not uncommon for the lesion to extend beyond the limits of the larynx and according to its situation infiltrate and invade the adjacent structures. This invasion may deform and displace the laryngeal structures and cause a rapid loss of laryngeal mobility.

We have had only one case of true cord paralysis. In this instance the left true cord was involved. A deformity or flattening of the lower portion of the ventricle was demonstrable on the roentgenogram. During roentgenoscopic study no change was noted in the ventricular outline during phonation. Pancoast describes three ventricular changes produced by cord paralysis: obliteration of lower half, posterior portion or entire ventricle.

Thickening of the retropharyngeal and retrotracheal tissues was a frequent complication in advanced tuberculosis of the larynx. There was similarly a marked bulging of the tissues due to cold abscess formation. This thickening must be differentiated from the swelling and thickening produced by other lesions. In children the retropharyngeal tissues are frequently swollen following cervical adenitis and upper respiratory infections. The periesophageal swelling following the impaction of a foreign body gives similar findings. In both instances the laryngeal structures are nearly always free from involvement. In 2 instances where a malignant growth arose from the pyriform sinuses, with extension into the cervical esophagus, we noted a marked thickening and swelling of the retrotracheal tissues. This swelling was localized to this region, in contrast to the generalized retropharyngeal and retrotracheal swelling noted in advanced laryngeal tuberculosis. Here again no involvement of the laryngeal structures was noted. In both instances there was noted a marked interference with swallowing and a jagged irregularity of the cervical esophagus when outlined with barium. The diagnosis of malignancy was made in both instances from the roentgen study and was not confused with laryngeal tuberculosis.

ANALYSIS OF FINDINGS

This series reported here represents 100 cases, 65 males and 35 females. As will be seen by a study of Tables I, II, and III,

TABLE I

LARYNGEAL INVOLVEMENT

		Minimal				Moderate				Extensive			
		Unilateral		Bilateral		Unilateral		Bilateral		Unilateral		Bilateral	
		Male	Female	M	F	M	F	M	F	M	F	M	F
Extent of pulmonary lesion in lobes	1					4	1						
	2		1		1	4	2	16	4		1	9	3
	3	2	2		1	2		3	4			3	1
	4				3			8				5	1
	5			1	4			4	3			3	3

(One negative chest in a male)

neither the quantitative involvements of the lungs nor the age of the pulmonary lesion has any bearing on laryngeal tuberculosis.

TABLE II

Duration of pulmonary symptoms *Months*	Minimal		Moderate		Extensive	
	M	F	M	F	M	F
3– 5			1	1	1	
5– 7			6		1	
7– 9	1	1		2		1
9–12		2	6	2	3	2
Years 1–1½		2	7	4	4	1
1½– 2	2	2	2	1	1	1
2–2½		1	5	2	2	
2½– 3				1	1	1
3–3½		1	4		4	
4 and over		3	7	3	5	1
Onset unknown			1			
No chest lesion			1			

TABLE III

QUANTITATIVE LESIONS NATIONAL TUBERCULOSIS CLASSIFICATION

Pulmonary Lesions

	Unilateral		Bilateral		Total		
	M	F	M	F	M	F	
Minimal	2	3	1	9	3	12	15
Moderate	10	3	31	11	41	14	55
Extensive		1	20	8	20	9	29
Negative					1		1
Total	12	7	52	28	65	35	100

TABLE IV

Type Pulmonary Lesion	Minimal		Moderate		Extensive	
	M	F	M	F	M	F
Negative			1			
Exudative productive			1		4	
Proliferative				1		
Caseous pneumonic	3	12	38	15	18	7

Qualitative analysis of the pulmonary lesions in the series (Table IV) shows 5 lesions of the exudative productive and 1 of the proliferative type. One case had a negative chest and 93 were of the caseous pneumonic type of tuberculosis.

ROUTES OF INFECTION

Tuberculous laryngitis is almost always secondary to pulmonary tuberculosis. A few cases of well authenticated primary lesions of the larynx have been observed (Coakley). The routes of infection may be by the direct route through the surface of the laryngeal mucosa from secretions coughed up from the lung, or indirectly through the blood and lymph. There is disagreement among authors as to the frequency of infection by these three routes. Wood, Thompson, Turner, and others, feel that the spread is nearly always by contact through the laryngeal mucosa, whereas Coakley gives the order of frequency as (1) through the lymphatics, (2) through the blood stream and (3) directly through the epithelium. In our series, the one with a negative chest (spine lesion) indicates a hematogenous involvement of the larynx. How many cases with pulmonary lesions are due to this cause is unknown.

The 5 cases with exudative productive lesions and the 1 of the proliferative type had little or no expectoration at any time and seldom had a positive sputum. The infection of the larynx may possibly have occurred through the blood or lymph and not directly from sputum containing tubercle bacilli, although this cannot be definitely excluded. Twenty of the cases had a complicating tuberculous lesion elsewhere in the body. Infection of the larynx by the lymphatic or hematogenous route is probably far greater than we suppose.

Study of the Gaffky count (Table V), which may show wide variations at different times, in no way helps to determine the route of infection but is interesting in itself. Three cases had low Gaffky counts,

TABLE V

Sputum examination Gaffky	Minimal		Moderate		Extensive	
	M	F	M	F	M	F
1				1	1	
2		1				
3	1	2	6			2
4		4	6		3	1
5			8	3	2	1
6	1	1	2	3	5	1
7	1	1	6	4	1	
8						
9			2	1		
10		1		1	1	1
Positive		1	8	1	5	1
Positive elsewhere—negative since admission		1	2	2	4	

13 had relative low counts and 9 had negative sputum.

CLASSIFICATION OF PULMONARY LESIONS

In our series of 100 cases of laryngeal tuberculosis, the majority of patients (93) had a caseous pneumonic or malignant type of pulmonary tuberculosis. Of these 93 (Table IV), 15 (3 males and 12 females) had minimal laryngeal lesions, 53 (38 males and 15 females) had moderately extensive laryngeal lesions; and 25 (18 males and 7 females) had extensive laryngeal lesions.

Of the remaining patients, 6 had benign types of pulmonary tuberculosis—2 with moderately extensive and 4 with extensive lesions of the larynx. One patient had no pulmonary lesion but showed a moderately extensive involvement of the larynx, with extralaryngeal extension and a marked thickening of the prevertebral soft tissues.

TABLE VI

	Minimal		Moderate		Extensive	
Laryngeal symptoms	M	F	M	F	M	F
Hoarseness	3	7	17	8	13	2
Hoarseness and dyspnea			1			
Hoarseness and dryness		1	5			
Hoarseness and pain		1	4	1	5	1
Hoarseness and aphonia at times			2	2		1
Hoarseness and tickling			2			
Hoarseness and dysphagia			1	1	1	1
Hoarseness, dryness and pain			2			1
Hoarseness, pain and dysphagia						1
Hoarseness, aphonia, pain and dysphagia			1			
Tickling			2			
Dryness			1			
Aphonia and dyspnea					1	
Aphonia and pain					1	
Aphonia and dysphagia			1			
Aphonia; post-diphtheria in childhood			1			
No symptoms		3	2	2	1	

LARYNGEAL SYMPTOMS

In our series, of 15 cases with minimal lesions, 3 were symptom-free; of 56 with moderate lesions, 4 were symptom-free; and of 29 with extensive lesions, 1 was symptom-free. In other words, 8 of our 100 patients had no laryngeal symptoms. Patton has pointed out that a number of cases with tuberculous laryngitis are symptom-free. Wood also finds that there may be no subjective symptoms in early lesions, because of which he contends that every patient with pulmonary disease should have an examination of the larynx.

Hoarseness was the predominating symptom (Table VI). It was the only symptom in 50 of the cases, 10 of whom fell in the minimal group, 25 in the moderate and 15 in the extensive involvements of the larynx. Hoarseness was present in association with other symptoms in 35 cases. Altogether it was a symptom in 85 of our 100 cases. The complaints with which it was associated were one or more of the following: dryness, pain, tickling, aphonia, dysphagia and dyspnea.

The hoarseness was a persistent symptom in 59 of the cases and an interval or inconstant symptom in 16. It was entirely absent in 7 patients who had other laryngeal symptoms, such as tickling, dryness, aphonia, dysphagia or combinations of these. One patient had an aphonia which dated from diptheria in childhood. One had a moderately extensive laryngeal tuberculosis and complained of no symptoms.

Summarizing, hoarseness was present in 85 cases, pain in 18, dysphagia in 7, aphonia in 9, dryness in 9 and tickling in 4. Two cases had dyspnea, due to laryngeal stenosis, and required tracheotomy. Laryngeal tuberculosis is always to be suspected in patients with pulmonary tuberculosis if hoarseness is present.

DURATION OF LARYNGEAL SYMPTOMS

Excluding the symptom-free group and the 6 cases wherein the onset of symptoms was not definitely known, no patient presented laryngeal symptoms for more than three and a half years (Table VII). Thirty of the patients had symptoms of three to seven months' duration; 59 presented symptoms for a year or less and 18 gave signs for one to two years. Of the one year group, 8 were minimal, 37 moderate and 14 extensive cases. Of the one to two year group, 3 were in the minimal class, 6 in the moderate and 9 in the extensive.

TABLE VII
DURATION OF LARYNGEAL SYMPTOMS

	Minimal		Moderate		Extensive	
	M	F	M	F	M	F
Months 0– 1		1	3	2	2	
1– 3		1	5	1	1	1
3– 5	2	1	4	1	3	1
5– 7		1	12	1	4	
7– 9		2	1	2		
9–12			1	4	1	1
Years 1–1½		1	3		5	3
1½– 2	1	1	2	1	1	
2–2½			2		2	
2½– 3			2		1	
3–3½			2		1	
4 yr. over						
Onset unknown		2	1	2		1
No symptoms		3	2	2	1	

TABLE VIII
AGE GROUP

	Minimal		Moderate		Extensive	
	M	F	M	F	M	F
Years 1–10						
11–20		3	8	5	2	2
21–30	2	6	12	9	6	2
31–40	1	2	9	1	8	3
41–50		1	5		6	
51–60			3	1		
61–70			2			
71–80			1			

AGE AND SEX INCIDENCE

Consulting Table VIII one sees that 61 of the cases occurred between the ages of twenty-one and forty; and 80 between the ages of eleven and forty.

Laryngeal tuberculosis occurs with about equal frequency in males and females below the age of twenty: 10 in males and 10 in females. Beyond the age of twenty, the lesion predominates in the male. The largest number of cases occurred between the ages of twenty and forty: 38 in males and 23 in females. Beyond the age of forty, laryngeal tuberculosis is not frequent; in the female it is rare.

SUMMARY

1. The roentgen study is an aid in the diagnosis of laryngeal tuberculosis; it should supplement the laryngologist's examination.

2. We obtain a sagittal view and can therefore gauge the height and width of the lesion as well as the presence of a subglottic extension.

3. Small intraventricular lesions which may be missed on mirror examination are demonstrable on the roentgenogram.

4. Small lesions situated deep in the interarytenoid area are not detected on the roentgenogram.

5. Roentgen examination gives a permanent pictorial record of the location, extent and progress of the lesion.

It is essential to have at least two roentgenograms of the larynx—one taken at rest and the other during phonation in addition to the roentgenoscopic study.

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VARIX OF THE PULMONARY VEIN*

By BENJAMIN H. NEIMAN, M.D.

CHICAGO, ILLINOIS

THE first case of pulmonary varix was reported by Hedinger at the Fifth Meeting of the German Pathological Society in 1907. He reported it merely as a rare condition not previously recorded in the literature. Since then 2 additional cases have been observed. The second by Nauwerck in 1923 and recently one by Klinck and Hunt.

Klinck and Hunt cover the literature on the subject of the pathogenesis of this condition quite thoroughly, discuss the various theories of the development of venous varices (Hasebroek, Forst, Bennett, Kaufmann, Jack and Lee) so that further comment would add little to our knowledge. Among these theories, the one referring to a congenital anomaly seems to be the most reasonable one. Klinck and Hunt include in their report, for the first time, a roentgenological study of this anomaly. In my case also, a complete ante-mortem roentgen study was made and the purpose of this paper is to correlate these findings with those found clinically and at the autopsy examination.

REPORT OF A CASE

On January 4, 1933, a white woman, aged fifty-seven, a native of Ireland, entered the Cook County Hospital stating that while she was washing herself, four days previous to entrance, her left arm suddenly dropped to her side and a marked weakness in her left leg appeared so that she had to drag it along. Upon questioning, she admitted that she expectorated and coughed a little and that her lips had been slightly blue for years. She had never had any hemoptysis but had a constant thoracic pain in the back and precordial region. She was subject to dizzy spells and occasional headaches. About twelve years ago, she was told she had gallstones but had been in fairly good health until the present complaints appeared. Three years ago, a shadow on the roentgenogram, at another hospital, was considered to be either a metastatic tumor of the lung from a hypernephroma or an aneurysm of the aorta.

Physical examination revealed an aged looking white female. The temperature, pulse and respiration were normal. Her blood pressure was 140/75. The lips and mucous membrane of the mouth were cyanotic. Examination of the lungs revealed equal expansion. There was an area on the posterior surface of the left side of the chest which had an increased tactile fremitus, dullness on percussion with bronchial breathing and numerous inspiratory râles. The heart borders were within normal limits and the sounds of fair quality with no murmurs. The left arm was paralyzed and sensation slightly impaired. A clubbing and cyanosis of the fingers and toes were noted. The motor power of the left leg was reduced and there was an exaggeration of the left patellar reflex.

Examination of the blood revealed, on entrance, Hb, 80 per cent; 5,310,000 red blood cells and 4,150 white blood cells. A later blood count showed Hb, 95 per cent, 5,000,000 red blood cells and 8,200 white blood cells, with a differential count of 76 per cent leucocytes and 24 per cent lymphocytes.

The urine was negative and the blood Kahn and spinal fluid findings were also negative. An electrocardiogram showed a slight left ventricular preponderance.

Because of the paralytic symptoms, a cerebral thrombosis was diagnosed. To account for the finding in the left lung, various diagnoses were suggested which included primary carcinoma of the lungs, congenital heart disease, in the form of a patent foramen ovale or transposition of the heart because of the cyanosis without cardiac murmurs. Nothing was found in the kidneys to corroborate the previous diagnosis of a hypernephroma. Roentgenoscopic examination revealed a well-circumscribed mass in the chest posterior and lateral to the heart without any pulsation visible. No apparent aneurysmal dilatation was made out. The pain in the left chest posteriorly became more severe and another roentgenoscopic examination revealed a circumscribed mass lying behind the left heart which seemed to be a part of the descending aorta. The aortic arch appeared normal. Barium in the esophagus showed a distortion of its normal passage, being pushed to

* From the Department of Pathology, Cook County Hospital, Chicago. R. H. Jaffé, M.D., Director.

one side. A pulsation of the mass at this time was thought to be visible. The cyanosis was considered to be due to the aneurysm pressing upon the pulmonary vessels producing a pulmonary stenosis and consequent vascular stasis. In a subsequent roentgenoscopic examination, Dr. Warfield diagnosed the mass as a benign neoplasm at the base of the left lung with no relation to the heart or large blood vessels.

Upon further questioning, it was learned that the patient began to notice a clubbing of the fingers about four or five years ago.

The clinical course was uneventful. The cerebral symptoms slowly disappeared but the patient gradually lost strength, finally dying about three months after her entrance into the hospital.

The autopsy, performed by Dr. R. H. Jaffé, revealed a marked clubbing of the fingers and toes; senile involution of the internal genitalia; focal, recent cortical encephalomalacia in the region of the right parietal lobe; moderate brown atrophy of the heart; diffuse bronchiectasis of the left upper pulmonary lobe; marked passive congestion of the spleen, liver and kidneys; slight compression atelectasis of the adjacent parts of the upper lobe and of the lower lobe by a huge varix of a branch of the second

Fig. 1. Roentgenogram of mass in the left upper pulmonary lobe. Note the smooth nodular periphery.

Fig. 2. Photograph of drawing of the mass in the left upper pulmonary lobe. Arrow leading from the varix into the left auricle. *L.U.L.* Left upper lobe. *V.* Varix. *L.A.* Left auricle. *L.V.* Left ventricle.

order of the pulmonary vein for the left upper pulmonary lobe with mural thrombi.

In the lower third of the upper lobe of the left lung, near the median aspect, there was a circumscribed, ovoid cavity 40×25×30 mm. The cavity was filled with soft, laminated, light purple-gray material and lined by a 1 mm. wall of a firm gray-white tissue. The upper median part of the sac was in open communication with a branch of the pulmonary vein. Dissecting the cavity revealed that it was part of a larger cavity occupying about half of the upper lobe. This cavity had many semi-spherical and ovoid outpouchings which were separated by thin, white, slightly transparent membranes. The pulmonary vein, with which the sac communicated, was a vein of the second order. The lung tissue surrounding the cavity was slightly compressed, dark purple-gray, but otherwise unchanged. The bronchi of the upper left lobe were diffusely dilated, the mucosa slightly injected. The interlobar fissure was completely obliterated.

The right lung was moderately distended and crepitant. On the sectioned surface it was purple-gray and moderately moist.

Histological examination of the cavity in the left upper lobe revealed the wall of the cavity to be on an average 0.5 mm. thick and to show the structure of the wall of a medium sized

vein. There was a very thin intima and a well differentiated media with regularly arranged, smooth muscle fibers surrounded by fine, collagenous and elastic fibrils. The external elastic membrane was often very prominent and presented itself as coils of compact tortuous fibers. The thrombotic masses were composed of hyalinized fibrin and disintegrated red blood

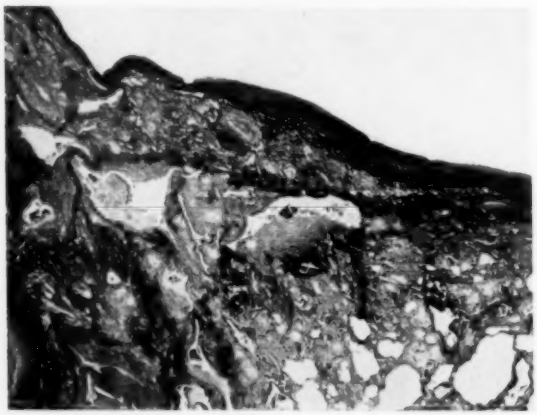

Fig. 3. Microscopic section taken through point of communication of varix with the pulmonary vein. Elastica stain. ×20.

cells with occasional small calcium concretions. On the inside, the old thrombus was covered by more recent deposits of fibrin, blood platelets and erythrocytes. At the base, a young and vascular granulation tissue was seen invading the thrombus. It contained many siderocytes and deposits of small, spherical calcium concretions. At the point of organization, the structure of the wall was less regular. There was more fuchsinophilic material between the muscle fibers and single blood vessels penetrated the media. Where ridges protruded into the cavity, a stretched and compressed bronchus could be seen between the opposite layers of the wall.

About the cavity, the lung parenchyma contained many small nodules composed of a peripheral zone of lymphocytes and a center of epithelioid cells in reticular connection. Inserted into this cellular reticulum were single multinucleated giant cells. In the region of the nodules, the elastic framework of the lung had been completely destroyed but elastica stains brought out, in some of the giant cells, tufts of fine fibrils which took the elastica stain. The nodules also showed relations to spherical, sometimes broken up calcium concretions of laminated structure. Some of the concretions were completely encased in giant cells.

The brain weighed 1,170 grams. The blood vessels at the base were thin walled with a few small hyaline plaques in the left half of the circle of Willis. The leptomeninges were markedly injected. The surfaces made by cutting were moderately hyperemic, especially in the region of the central ganglia, which were discolored purple-brown. The microscopic examination of the right parietal lobe showed the cytoarchitecture of the cortex well preserved. The Nissl substance stained well. There was a diffuse capillary congestion and an increased satellitosis. In the deeper portions of the cortex, circumscribed areas stood out because of an extreme capillary congestion which was associated with a swelling and proliferation of the adventitial cells. In these areas, the ganglion cells showed severe alterative changes. The Nissl bodies had completely disappeared and the cytoplasm was pale and homogeneous. The nuclei, which were often displaced to the periphery of the cell, were more or less shrunken, the nucleoli were less conspicuous than normal and the nuclear membrane was thickened. There were many glia cells in these areas, the majority of which were of the Hortega type. In the region of the central ganglia, some of the ganglion cells were vacuolated and had lost their Nissl bodies. There was a marked diffuse capillary hyperemia.

DISCUSSION

Hedinger accounted for the presence of a duodenal ulcer in his case on an embolic basis while Nauwerck attributed a transient paresis in his on the same phenomenon. In my case, the patient entered the hospital because of a sudden paresis of the left arm and left leg. This paresis was apparently on the basis of an angiospastic, focal encephalomalacia and not embolic since clinically a disappearance of the symptoms was observed and no emboli could be demonstrated in the cerebral arteries.

A point of interest, which Nauwerck does not mention, is the presence or absence of a hypertrophy of the right heart. The thickness of the right ventricle in Hedinger's case was 3 mm. while in that of Klinck and Hunt it was 9 mm. In my case there

was no hypertrophy of the right ventricle; its wall was 3 mm. in thickness. In view of the cyanosis and clubbing of the fingers and toes, one could expect a hypertrophy of the right ventricle. The absence of this hypertrophy may have been due to the fact that the myocardium did not possess sufficient reserve power to hypertrophy, throwing its load upon the peripheral vascular tree. Or, since the varix is apparently congenital, the circulation may have adapted itself so as not to throw any abnormal load on the heart.

Clinically all the four cases had signs and symptoms which pointed towards an intrapulmonary lesion. Hedinger's and Nauwerck's cases both had hemoptyses which were of an intermittent type. Klinck and Hunt's and my case had cyanosis with some difficulty in breathing. The physical signs in the latter two cases were indicative of intrathoracic disease. Obviously a differential diagnosis based on such meager evidence would include a formidable array of conditions. A roentgenological study, however, eliminates many, leaving only a few to choose from. Unfortunately the two early cases had no roentgen examination, but a comparison of the roentgen shadow in my case with that of Klinck and Hunt shows a practically identical finding. The fact that no cases were reported prior to 1907 and none have been diagnosed ante mortem, suggests that these cases occur but escape clinical recognition. Although I have only one other case to compare roentgenologically, still it helps to form a basis of diagnosis.

The roentgen examination of the chest in my case revealed a well circumscribed, lobulated tumor-like shadow of even density occupying the upper lobe of the left lung. This shadow fused with the heart and diaphragm in the posteroanterior view of the chest. Roentgenoscopically it was possible to separate this tumor from the heart shadow and to show that it had no relation to the ascending aorta. These, together with the clinical findings, and the fact that comparison with the roentgenogram taken three years ago showed no change in size or shape, suggested a benign tumor of the lung. That the condition was not a lung abscess was evidenced by the lack of clinical findings of leucocytosis and fever and roentgenological findings, which will be shown later, to substantiate such a diagnosis. The mass was too well circumscribed and of too long a duration to be a primary carcinoma of the lung. It was not in the mediastinum at roentgenoscopy, therefore a mediastinal tumor could be excluded. Pericarditis with effusion was ruled out since the mass had no relation to the heart. The mass was not connected with the aorta and there was no erosion of the spine or ribs; thus a saccular aneurysm of the descending aorta could be eliminated.

Benign tumors of the lung are relatively rare. Hickey and Simpson, in a review of the literature, report 40 cases, which at post mortem or by biopsy were chondromas. In our material, in a series of over 5,000 autopsies, 3 cases of benign tumors of the lung were found, 2 of which were chondromas and 1 an adenoma arising from a bronchus, while primary carcinoma of the lung was present 72 times.

The diagnosis of these neoplasms during life, as Hickey and Simpson point out, is dependent upon an accurate roentgenological interpretation. Physical signs have been of little aid in differentiating the few larger tumors from other intrathoracic disease and in many instances careful percussion and auscultation gave no evidence of tumor.

Since the shadow that the pulmonary varix casts on the roentgenogram is sharply circumscribed in character, it serves to distinguish it from those conditions which are of an inflammatory genesis. Tuberculous lesions and abscesses may be distinguished from this condition because of the presence of the irregular peripheral inflammatory infiltrations in the former.

The two conditions which resemble pulmonary varices closest are chondroma and solitary echinococcus cyst of the lung. Solitary echinococcus cyst of the lung is

more commonly encountered than primary pulmonary chondroma and the roentgenographic appearances are quite similar. Pulmonary varices resemble both and are even more uncommon so that errors in interpretation are easily understood, especially if one is influenced by the law of probability. However, since echinococcus cysts and chondromas are the most common benign tumors of the lung, the problem of diagnosis would rest in differentiating these conditions from pulmonary varix.

As regards the periphery, the pulmonary varices, as seen at post mortem in all the cases and roentgenologically in the last two, were invariably nodular. The chondromas are similarly nodular, even though these irregularities may be slight, while the echinococcus cyst is either perfectly smooth or presents slight inflammatory infiltrations in the immediately adjacent lung tissue. Frequently present in chondromas is the patchy, irregular distribution of lime salts, due either to simple calcification of the cartilage matrix or to true ossification. Calcification of the laminated chitinous lining membrane of the echinococcus cyst is occasionally present but is a more uniform process. In pulmonary varices it has been observed in very small amounts.

If the nodular periphery is present on the roentgenogram together with symptoms of cyanosis, dyspnea, cough, clubbing of the fingers or toes, and intermittent hemoptyses, with an absence of serological and clinical evidence of echinococcus infestation, one should think of a pulmonary varix.

SUMMARY

To the three previously reported cases a fourth case of pulmonary varix is added. None has been diagnosed ante mortem. As an aid in diagnosis, roentgenological study is advocated to differentiate it from other benign tumors of the lung, especially solitary echinococcus cyst of the lung and primary pulmonary chondroma.

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FIBRIN BODIES IN THE PLEURAL CAVITY*

WITH REPORT OF THREE CASES

By ARNOLD SHAMASKIN, M.D., *and* JACOB ROGOFF, M.D.
BEDFORD HILLS, NEW YORK

ROENTGENOGRAMS occasionally present unusual shadows which are puzzling. Even if only medical curiosities, they must be readily recognized as such by the roentgenologist in order that he may differentiate the merely abnormal from the pathological.

With this point in view we wish to report 3 cases of fibrin bodies in the pleural space, which we encountered recently. Although the foreign literature is fairly replete on the subject of fibrin bodies in the pleural cavity, the American literature, as far as we are aware, deals very inadequately with this matter. Fleischner[1] in 1922 was apparently the first to recognize and describe a freely movable body in the pleural cavity of a patient with pulmonary tuberculosis, who, during the course of pneumothorax therapy, developed an effusion. He writes that "during deep inspiration the level of the fluid was lowered and there appeared above the surface of the effusion a sharply defined, rounded, homogeneous shadow, the shape and size of a silk cocoon, whose position in the pleural cavity could be changed by shifting the patient from side to side, and whose specific gravity was apparently higher than that of the surrounding fluid." He concludes, "that since small fibrin flakes are commonly found in pleural exudates, the conglomeration of these to form larger fibrin bodies should be possible."

Düll,[2] Maendl,[3] and Poindecker,[4] in 1925 reported 3, 2 and 1 cases respectively. The first mentioned believes that these masses arise from blood fibrin that was separated out in the pleural cavity from the blood after injury to an intercostal vessel during a refill. The latter two are of the same opinion as Fleischner regarding the origin of these bodies.

Stöffel,[5] in 1926, reported 1 case, which later came to autopsy. In his case the fibrin body was found after the disappearance of an effusion. He believed that fibrin was formed around a nidus of some sort, possibly an adhesion which had been torn during a refill. He compares the formation of these fibrin bodies to the formation of calculi in the gallbladder. At post mortem this mass resembled caseous material and was yellow in color. Microscopically it contained no blood cells or tubercle bacilli and consisted solely of fibrin.

In 1927 Mendl[6] reported 3 cases, 2 of which had freely movable fibrin bodies and the third was stationary.

Lossin[7] in the same year reported 1 case in which the fibrin body was adherent to the parietal pleura.

In 1928 Pomelzoff[8] reported the case of a patient who died of a spontaneous pneumothorax and heart failure following shortly upon a pneumothorax refill. The patient came to necropsy. A mass, the size of an English walnut, yellow-gray in color and firm, was found firmly adherent to the parietal pleura in the costophrenic sulcus. Histologically and chemically it consisted of pure fibrin.

In this country the only case reported was by Ballon[9] in 1929, who observed a fixed fibrin mass in the pleural cavity of a patient who had a hydropneumothorax.

CASE REPORTS

CASE 1. S. B., male, aged thirty-seven. Admitted March 20, 1931. History of the present illness dates back seventeen years. It began with hemoptysis which recurred at intervals up to admission. Physical and roentgenological

* From the Montefiore Hospital Country Sanatorium, Bedford Hills, New York. Presented at a Monthly Conference of the Tuberculosis Division of the Montefiore Hospital for Chronic Diseases and its Country Sanatorium, December 28, 1933. Dr. Maurice Fishberg, Chief of Division.

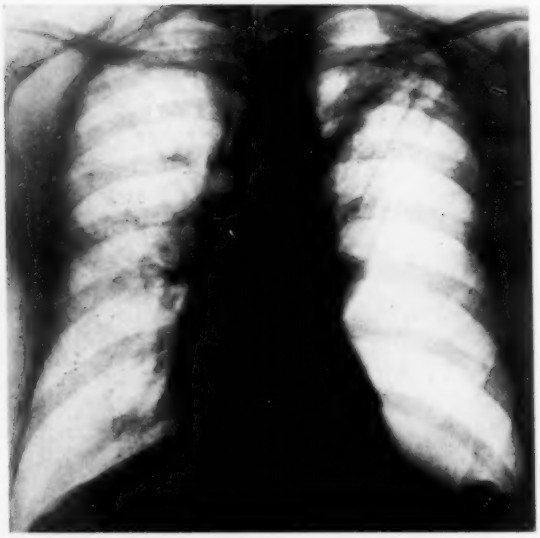

Fig. 1*a*. Case I.

findings revealed a productive lesion involving the apex of the right lung and the entire left lung with a large cavity in the infraclavicular region. Both domes of the diaphragm were normal. On account of symptoms and signs of progression, pneumothorax therapy was instituted in September, 1931. In July, 1932, an effusion developed. When in the course of time the fluid became almost absorbed a rounded shadow was observed resting on the outer half of the dome of the diaphragm (Fig. 1*a*). Subsequently fluid reaccumulated in the left pleural space and the above described shadow became invisible (Fig. 1*b*). In order to determine whether the above described shadow was absorbed or merely submerged, a roentgenogram of the chest was taken while the patient was lying on the right side with shoulders lower than the hips (Fig. 1*c*). The film revealed again a rounded, sharply demarcated shadow, the size and shape of a hen's egg, apparently attached to the middle part of the diaphragm with adhesions beginning to form between it and the chest wall bridging the costophrenic sulcus.

Case II. A. Z., male, aged forty-three, admitted February, 1933. He gave a history of two previous admissions to another sanatorium. On the second admission a fibrocaseous lesion affecting the middle of the right lung with a cavity was found. Both domes of the diaphragm were normal. Pneumothorax therapy was instituted in June, 1932. In December, 1932, an effusion developed. On admission to this sanatorium February, 1933, the right lung was almost completely collapsed by a pneumothorax extending from base to apex with a trace of fluid in the costophrenic sulcus. An oval, sharply defined, smooth-edged shadow, about the size of an English walnut, was seen resting on the outer part of the dome of the diaphragm. Another shadow of lesser density, also rounded and sharply demarcated, was apparently attached to the parietal pleura just above the costophrenic sulcus (Fig. 2*a*). A roentgenogram taken December 16, 1933 (Fig 2*b*) showed that the pleural space

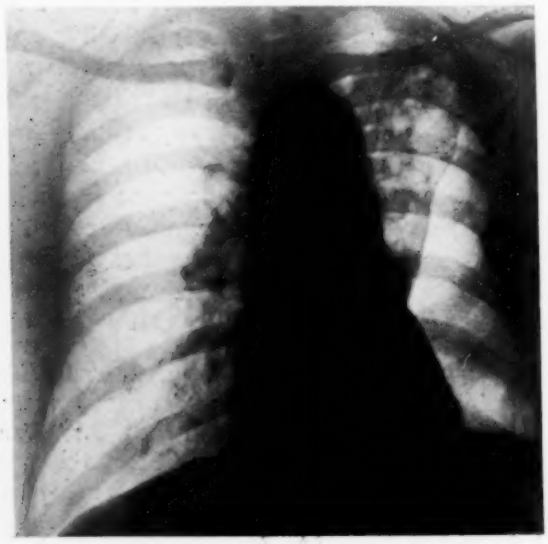

Fig. 1*b*. Case I.

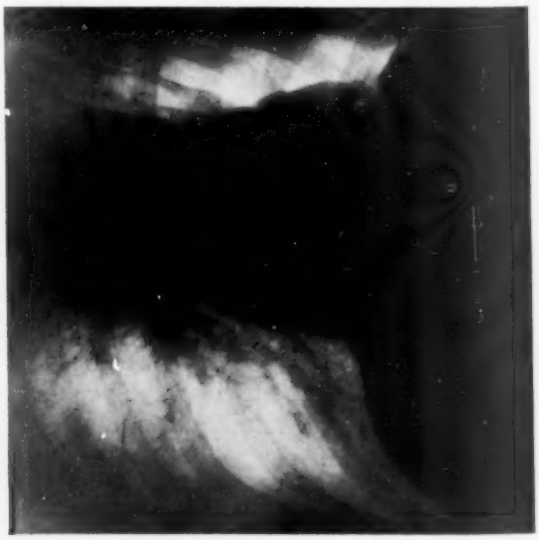

Fig. 1*c*. Case I.

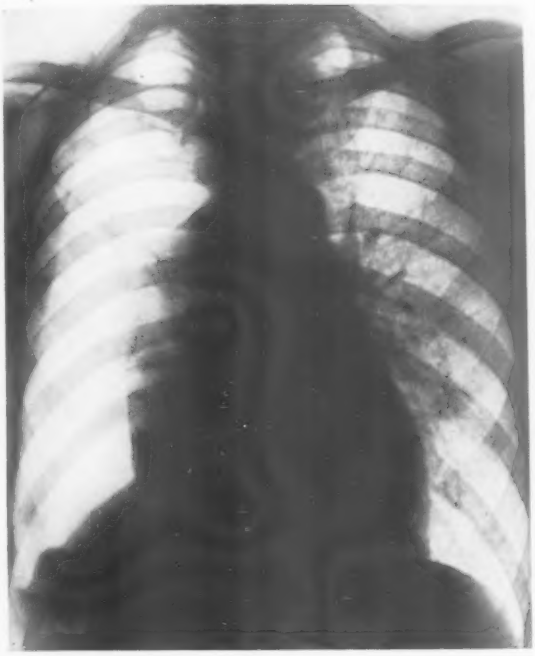

Fig. 2*a*. Case II.

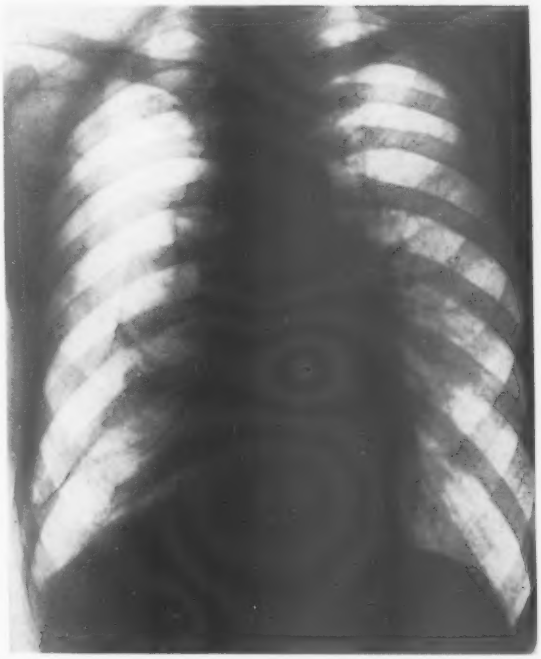

Fig. 2*b*. Case II.

began to obliterate from below and the above described shadows had disappeared.

Case III. L. M., girl, aged sixteen. Onset of present illness March, 1932, with cough and fever. Pneumothorax was induced on the right side in August, 1932. Admitted January, 1933. On admission a partial pneumothorax was found on the right side extending from base to apex and collapsing principally the upper third of the lung. A giant cavity in the mid-zone remained uncollapsed. A roentgenogram taken February 2, 1933, revealed an effusion covering the dome of the diaphragm. Another roentgenogram taken August 30, 1933 (Fig. 3*a*) revealed the fluid almost absorbed and an elongated shadow with smooth, sharp edges somewhat ir-

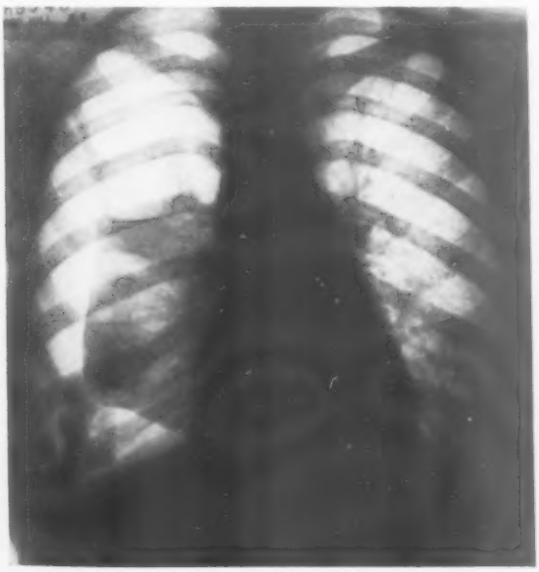

Fig. 3*a*. Case III.

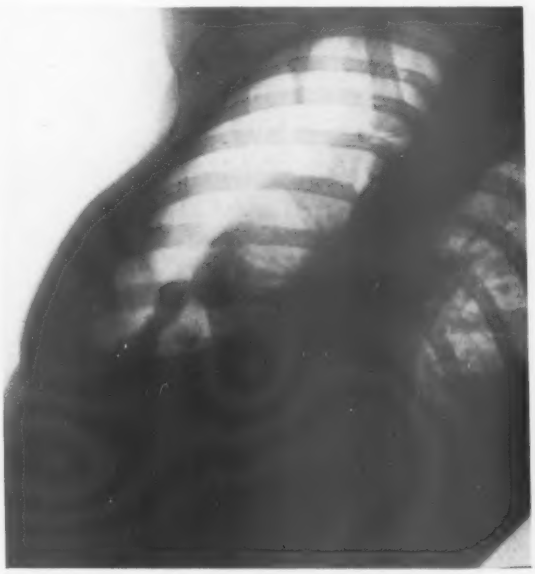

Fig. 3*b*. Case III.

regular in outline extending from the tip of the partly collapsed lower lobe to the dome of the diaphragm. A roentgenogram taken December 26, 1933 (Fig. 3*b*) with the patient leaning to the left showed the above mentioned shadow more sharply outlined and on roentgenoscopic examination the lower end was found free.

CONCLUSIONS

From our own observations and from a survey of the literature, the following generalizations may be drawn concerning fibrin bodies in the pleural space:

1. These fibrin bodies have no clinical significance.

2. They are found in cases of hydropneumothorax and become visible when the level of the fluid drops.

3. They may be either free in the pleural space or attached to the pleural surface.

4. They usually disappear without leaving a trace. They may, however, remain in the pleural space and calcify eventually.[8]

5. Their average size is that of an English walnut and they are usually homogeneous, smooth in outline with sharply demarcated edges.

6. They may be confused with neoplasm, encysted empyema, exostosis of a rib and foreign body.

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THE PROGNOSIS OF FRACTURES OF THE VERTEBRAE

By E. WALTER HALL, M.D.
DETROIT, MICHIGAN

THE propriety of a roentgenologist discussing the prognosis of vertebral fractures may be open to question. He is, it is true, concerned with the case chiefly in a diagnostic capacity and is directly concerned with only the morphologic features of diagnosis. However, he is privileged to examine many patients immediately after injury as well as after treatment and to observe the end-results of various methods of treatment by different surgeons. If he is alert to his opportunities he correlates roentgen observations with information obtained from the patient as well as from discussions with the clinician. Such observations form the basis for judgments which are impersonal and unbiased. The views expressed here are the result of such observations and of a special study of 155 consecutive cases of vertebral fracture.

Prognosis is at best an indefinite subject. Statements concerning prognosis must always be modified by provisos as to the nature of the insult, the status of the individual, the treatment, and the presence or absence of intercurrent factors. For the sake of definiteness we shall therefore limit this discussion to fractures of the spine which do not involve questions of paralysis or cord injury, or obvious predisposing diseases such as malignancy.

There exists both in the minds of the laity and of the physician, generally speaking, a very imperfect idea of what to expect from spinal fractures. Even in the literature, which up to the last few years dealt almost exclusively with fracture dislocations, these being the only ones usually recognized, there exists a wide variance of opinion. For example, in the case of fractures of the lumbar body we have the opinion of Kessler, who allows a permanent disability of 50 per cent or over, as contrasted with that of Jimeno-Vidal who believes properly treated cases recover completely after four or five months. Campbell, Forrester, Scudder, and other orthopedists, allow one to two years for treatment with support and are very pessimistic about permanent recovery. We are interested to know whether the prognosis of vertebral fractures can be put upon a more logical and reasonable basis, free from the old "broken back" tradition, with the mental element properly evaluated.

I. FRACTURES OF THE VERTEBRAL BODY

Fractures of the vertebral body are of two main types, there being either fragmentation or bone condensation. Both types involve the upper surface of the body, and though in severe fractures fragmentation may extend to the caudal surface, we have found no case of simple compression in which the inferior surface or cartilage plate was injured. There may or may not be displacement of body fragments and when this occurs it is generally slight and usually anteriorly. As in fractures elsewhere, there is always some tearing of soft tissues with extravasation of blood and lymph. There may be fracture of the superior cartilage plates with possible escape of the nucleus pulposus. In our series about 10 per cent of the uncomplicated body fractures were multiple, though in multiple fractures there should be a strong suspicion of underlying pathological conditions. Space does not permit the discussion of all the complicating factors, so we pass on to a consideration of the eventualities of healing of a fractured vertebra. From the morphologic standpoint there are several possibilities, two or more of which may be evident in the study of the roentgenogram of a single healed fracture.

1. *Complete healing with no residual disturbance in structure or contour.* This is at present rare, in fact we have seen no case in which a good roentgenogram does not reveal a slight residual bone change. How-

ever, as the surgeons are now using earlier and more efficient reduction and immobilization, we expect to see many such cases in the future with early and complete restoration of function.

2. *Healing with slight deformity of the body and preservation of the integrity of the intervertebral discs.* This is, in our series, the most common type, comprising 39 per cent of the total. There is no important change in vertebral alinement, no evidence of degenerative change and no roentgen basis for symptoms or disturbed function. The large number in this group speaks well for the treatment methods of the attending surgeons.

3. *Healing with increased deformity but preservation of the cephalic disc.* This is the neglected case or the case treated without proper reduction. We believe it is *seldom* due to *too early* removal of support. We are inclined to agree with Jimeno-Vidal that three to five months is sufficient for the healing of even cancellous bone and that further fixation is injurious rather than beneficial, though many men still use fixation for nine months to two or more years. Incidentally, if sufficient attention is paid to the general condition of the patient, with physiotherapy, exercise and a general "building up" routine, the soft tissues should also be completely healed by the same time.

We may state in this connection that in our opinion the so-called Kümmell's disease or rarefying osteitis without fracture which has attained a place in so much of our literature is merely due to the further compression of unrecognized fractures which are improperly treated. Certainly if so-called concussion or disturbance in blood supply were responsible for "rarefying osteitis" few compression fractures would have a chance for the satisfactory healing that usually occurs. We have never seen a case of this condition that seemed satisfactorily proved but such is the weight of tradition that every writer seems called upon to discuss it, though disparagingly. In considering prognosis of vertebral injuries "Kümmell's disease" may therefore be disregarded.

These rather marked vertebral deformities with intact cartilage comprised only 10 per cent of our group. They are usually associated with ligamentous ossification.

4. *Healing with ankylosis.* In 23 per cent of the series there was formation of a bony bridge from the injured body to its cephalic neighbor. In 2 cases there was also bridging to the inferior vertebra. With bony ankylosis precluding motion there should be no pain at the fracture site. The spine should be even stronger though less mobile than before the injury, and as the spine is primarily a supporting structure and free mobility is not an important function except in the case of contortionists or acrobats, this should not be a basis for disability. Even in the cervical spine where mobility is of more importance than elsewhere, many persons have gone through life unconscious of any limitation from congenital fusion of two vertebrae.

Some of these cases show a kyphos or moderate lateral angulation. Extreme angulation is unusual in uncomplicated cases. We believe slight variations in alinement are overrated as bases for disability. We have all seen "hunch-backs" doing heavy manual labor and there are many cases of scoliosis or kyphosis of considerable degree which are symptomless and do not incapacitate to an appreciable extent. The spine readily compensates for even such a marked deformity as an intercalation and this is usually discovered accidentally and not because of symptoms.

It has been stated that after production of new bone is complete the patient should be symptom-free. We believe this is an arbitrary, though perhaps workable, rule to apply in compensation cases. The production of new bone is not in all cases accompanied by symptoms; in our experience this is seldom the case. In our routine films preliminary to gastrointestinal studies we find many spines showing marked hyperostosis without apparent associated pain. The fracture patient may therefore be

symptom-free long before reparative or irritative changes due to changing stress and strain are complete.

5. *Healing with disturbance in the cephalic disc.* In only 6 per cent of our series was the disc importantly narrowed or distorted. In no case was there complete destruction or complete absorption of cartilage. This would be a plausible basis for symptoms providing there was motion, but in the majority of cases narrowing and irregularity of the disc was found to be associated with bony ankylosis which should render it symptomless.

In 20 per cent of the series, an unexpectedly large number, there was definite evidence of fracture of the superior cartilage plate with herniation of the nucleus pulposus into the injured body. This is often considered a basis for symptoms. When nucleus hernia was first resurrected it was hailed by many as a cause of back pain. Having now observed even multiple hernias in patients comparatively symptom-free and without history of trauma, we no longer give it an important place. In one-third of our cases there was a protecting bony ankylosis.

The question of pressure on the roots of the spinal nerves frequently arises as an eventuality of vertebral body fracture. One author refers to the frequency with which typical girdling pains or parasthesia occur in cases of fracture of the processes or bodies. These symptoms, which are characteristic of pressure on a nerve, are peculiarly absent from the patients we have observed and in consultation with others who see many fractured spines we obtain reports of a similar rarity. Unless cord or pressure symptoms are present early, we do not believe they are late sequelae.

Only 2 cases of our series showed marked calcification and ossification in the paraspinous soft tissues. This would seem to indicate that products of injury such as hemorrhage and edema are early absorbed in nearly all instances and would also suggest early repair of the soft tissue damage.

The study of this series of vertebral body fractures leads us to conclude that in the majority of cases, namely those with little or no vertebral deformity, no narrowing of the discs, or with fixation by bony ankylosis, approximating 70 per cent of all our vertebral body fractures, the ultimate prognosis should be good both for recovery of function and freedom from symptoms. This percentage should be materially increased by the application of the newer treatment methods of Dunlop and Parker or Jimeno-Vidal, especially the latter. With excellent results, he has been using complete reduction in marked hyperextension, application of a cast in this position and institution of physiotherapy and exercise so that patients are actually in better physical condition at the time of removal of the cast than previously. He limits the fixation period to from twelve to twenty weeks, depending on the severity of the trauma and unfavorable factors, and eliminates all supports on removal of the plaster. He states that a surgeon who gives his patient a brace after the plaster is removed does him irreparable harm and makes a chronic invalid of him.

There is much to be said for the last statement. Psychotherapy plays an immense part in the prognosis of spinal injuries. Like all other adults, patients who have had spinal injuries are subject to back pain, but naturally their back pains are attributed to their fracture. By the same token if a patient has back pain and a roentgenogram discloses a deformed vertebra, *that* is too often immediately held responsible. We would emphasize the fact that the patient who has had a fracture deserves as diligent a search for disturbances in nutrition and elimination, for focal infection or endocrine dyscrasia, as does the individual who has not had trauma.

In the case of the so-called delayed or recurrent symptoms not continuous with the period of early disability, one should be especially careful to consider malingering, psychosis, or intercurrent cause of pain. The patient's complaints are therefore not to be taken too seriously. Unfortunately

for statistical purposes the large majority of our series are compensation or public liability cases with monetary considerations affecting prognosis. About 5 per cent are free from such taint and also entirely able and symptom-free according to their own statement.

We have in our files records of a patient who sustained a vertebral body fracture, obtained an excellent result with fixation—in this case by an Albee graft—and did heavy work for several years thereafter. All this despite the fact that he had a moderate vertebral body deformity, a loss of the forward convexity of the lumbar curve and a marked reduction in the normal dorsal convexity with, of course, limitation of mobility due to the graft. This case also illustrates another point under discussion, for he recently complained of low back pain, which he attributed to the old healed fracture, and was re-examined. This study revealed no basis for symptoms from the fracture but did show typical lumbosacral disease with narrowed disc, lipping, and sclerosis.

Another case which illustrates a not uncommon type is that of a man who was examined for rather recent low back pain incident to injury. Roentgenograms did reveal transverse process fractures but in the course of the complete study which is our rule we also showed a wedged sixth thoracic segment. This was smooth in contour with intact discs, and had every appearance of compression fracture, the symptoms of which had been so slight that it remained undiscovered. Wedge-shaped bodies are frequently discovered in this area. They are often wrongly diagnosed as congenital, the diagnostician overlooking the fact that vertebrae do not develop in such a manner as to make simple anterior wedging possible. This represents a large group of cases who in pre-roentgen days had fractures, considered them as "sprained backs," were incapacitated for a few weeks, if at all, returned to heavy duty, and were much better off than many workmen who have had the benefits of modern roentgenography and poor psychotherapy. Disease such as epiphysitis during the period of growth is a frequent cause of narrowing of the anterior parts of bodies, but this is rarely if ever confined to a single segment.

Then there is the case of a physician who sustained a compression fracture in an automobile accident—no money involved. There was minimal deformity but there was definite rupture of the cartilage plate and herniation of the nucleus pulposus. Despite somewhat neurotic tendencies he is not in the least incapacitated. It took strong psychotherapy coupled with good surgical treatment to produce this result. Contrast this case with that of another patient who had a rather severe fracture in the lower cervical region, but obtained excellent healing with firm ankylosis, making the spine actually stronger than before and with no slightest possibility of displacement. After four years it is impossible to wean him from an orthopedic collar. Perhaps a few hundred dollars monthly disability insurance has some bearing on his "disability."

We believe it the duty of the orthopedists, industrial surgeons and roentgenologists to educate the public and the profession to the idea that uncomplicated fracture of a vertebra is not a hopeless and often not a serious accident judged from the standpoint of ultimate prognosis, and that the person who suffers a fracture of a spinal segment has as much right to expect a good result, symptom- and disability-free, as had his fracture been of the Colles or Pott type. We consider the prognosis of vertebral body fracture to be decidedly better than that of the os calcis, the head of the tibia and similar lesions, provided the psychic element is eliminated.

II. PROGNOSIS OF TRANSVERSE PROCESS FRACTURES

Our series of spinal fractures showed 39 per cent of the total number to involve the lumbar transverse processes, proving that this is a common lesion, though occasional cases are still being reported as unusual. The idea of their rarity, repeated in one of our latest orthopedic volumes on the spine, is a heritage from pre-roentgen days when

they went unrecognized and untreated. Our series showed about equal involvement of the right and left sides and slightly more than half of the cases showed multiple fractures.

There is disagreement among authors as to the prognosis of this type of vertebral fracture. Campbell allows immediate total disability of six weeks (i.e., while on a Bradford frame with traction), and partial disability during the wearing of a brace for from three to six months with possible permanent symptoms in untreated cases. Kessler allows from three to seven months' temporary disability and from 10 to 25 per cent permanent disability. Steindler allows five to six weeks for bed rest and gives 10 per cent disability for three to six months, while Kennedy states that most cases should be symptom-free and working in from two to three months. These fractures are, in our opinion, minor injuries. We consider the bone injury secondary in importance to the soft tissue injury and would govern the prognosis and treatment accordingly. The function of the processes is to serve as attachments for soft tissue structures and the prognosis is only that of torn soft tissues. Disability should be determined by the time necessary for the formation of fibrous tissue, and should be measured in weeks rather than months. The fibrous tissue formed is actually stronger than the elastic tissues it repairs. Whether the small fragments unite or not is a matter of small consequence. The prognosis is independent of the severity of the bone injury. Cases with wide separation and therefore with only fibrous union have no more symptoms than those cases with merely a subperiosteal crack. Forrester reports a case where all the lumbar processes were fractured and in which the patient returned to heavy work in thirteen weeks having had only adhesive strapping, and a case of minor injury to one process immobilized by a cast with a disability of thirteen months. His advice is that the less treatment given the better for the patient and that no case should have plaster or braces, but merely adhesive strapping, and all should return to work within three months. While a body cast may at times be permissible in the treatment of severe injuries in which transverse processes are involved, since it provides greater fixation, it has, except in the hands of a very wise and tactful surgeon, a tendency to make the patient overrate his illness and have a longer disability. In most cases adhesive strapping is as effective as in rib fracture and it certainly is better for the mental attitude of the patient.

We believe the prognosis of transverse process fractures is better than that of rib fractures and as in the case of the vertebral body, if symptoms are unduly prolonged or delayed, malingering, psychoses, or general causes of back pain rather than the fracture itself should be blamed. The surgeon who treats a simple lesion by heroic measures may do irreparable harm to his patient.

CONCLUSIONS

Fractures of the vertebral bodies and transverse processes have been overrated as bases for disability, largely due to poor psychotherapy and improper treatment.

Over-treatment has been responsible for more bad results than under-treatment.

A study of roentgenograms and clinical observations of a series of 155 cases of healed fracture reveal little morphologic or anatomical basis for disability in the great majority of the series.

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MULTIPLE SPONTANEOUS IDIOPATHIC SYMMETRICAL FRACTURES*

By LOUIS A. MILKMAN, A.B., M.D.

Director, Department of Roentgenology, Scranton State Hospital and Mercy Hospital
SCRANTON, PENNSYLVANIA

THE purpose of this paper is to report what I consider a new disease entity: multiple spontaneous idiopathic symmetrical fractures. The name is descriptive and until its etiology is determined, a shorter name cannot be given.

In this paper is a complete case report, including serial roentgen examinations and complete autopsy findings.

Multiple spontaneous idiopathic symmetrical fractures is a systemic skeletal disease which involves the entire skeleton, the flat as well as the tubular bones. Its origin is in the cortex of the bone and it slowly encircles the entire bone extending into the marrow cavity.

Its special peculiarity is the symmetry of the involvement of the paired bones. It may also be symmetrical as to direction and location of the lesions. The disease is progressive with periods of intermissions and exacerbations. The onset is insidious. The etiology is unknown. The great number of fractures which may occur is surprising. The symptoms of this disease are: A disturbance in the gait which becomes a waddling type of motion. There is pain in the lower back and extremities, also localized pain in the individual bones before there is any physical or roentgenological evidence of disease.

The physical examination, except for the tenderness over the skeletal tissues, is grossly negative. There is no crepitus or other abnormality of the involved bones.

The roentgen appearance of the lesions is characteristic. There are bands, or zones, of increased radioparency. The bone appears as if the entire bony structure had been erased at the point of involvement. These zones vary in size from a few millimeters to a centimeter. The bone above and below the transparency zone looks normal, except for a thinning of the cortex and increased striations of the spongiosa, as well as a moderate deficiency of the calcium content. The edges of the bones are irregular. The transparency zones have lost all of the calcium and are composed of structureless supportive or ground substance. At the onset of the involvement of a particular bone, a small, round, or irregular punctate area of calcium deficiency is seen in the cortex and this gradually increases, extending across the bone transversely to its long axis. One bone may be involved in several areas and when the lesion is complete, a transparency zone or band of increased radioparency appears. There is no evidence of bending or bowing of the bones; there is no deformity until late in the disease when complete separation, displacement and overlapping of the fragments may occur. The transparency zones are increased in number and width. Atrophy of the bone, while fairly pronounced, is secondary to this disease process and is partly due to the limitation and disturbance of function of the involved bones. Another peculiarity of this disease is that there is no attempt at repair, no evidence of callus formation, no signs or evidence of absorption of the fragment ends. They remain irregular and jagged. There may be slight periosteal reaction but the periosteum does not become calcified.

The varied localization, vague pains, the atypical disturbance of motor function, the absence of physical findings, the normal blood chemistry as to urea, uric acid, blood calcium and phosphorus, have resulted in a variety of diagnoses for this condition. Clinically, it has been diagnosed as rheumatism, arthritis due to focal infection, sacro-

* Read before the American Congress of Radiology, Chicago, Ill., Sept. 25–28, 1933.

iliac sprain, neurosis, dystrophia myotonica and osteomalacia. After the roentgen study was made, the following diagnoses had to be considered: fragilitas ossium, late rickets multiple myeloma, hunger osteopathy, disturbance of the parathyroid glands and other endocrine glands.

This disease, with its peculiar symmetrical skeletal involvement, with its vague symptomatology, early absence of physical findings, is a separate and distinct disease entity which has heretofore been grouped with late rickets, osteomalacia, osteopsathyrosis or fragilitas ossium and hunger osteopathy.

CASE REPORT

Miss G. M., aged forty-three, school teacher.

History of Present Illness. In June, 1925, due to lameness in the back, pain on stooping over, difficulty on arising from either the lying or sitting posture, also pain across the small of the back, the patient sought medical aid. She was referred by her family physician to a gynecologist, orthopedist, nose and throat specialist and neurologist.

In 1926, because of no improvement in her condition, she was studied at Clifton Springs and after a stay at the sanatorium, she was slightly improved.

In 1927, because of the return of her symptoms, she was studied at the University of Pennsylvania by Dr. Spiller and his associates. On her return from the hospital, with her condition unchanged, she was under the care of her family physician.

In 1928, her family physician referred her to a clinic given by Prof. Bauer, of Vienna, and she gave the following history: In addition to the previous complaints, her condition is growing worse. She formerly was able to walk to school daily, now she cannot walk because "as soon as she steps on her right leg, she feels a severe pain in the upper end of the femur. There is also a feeling as if her legs were going to give way."

In 1929, her condition did not improve, the same symptoms were present, but were exaggerated.

In 1930 and 1931 new pains developed in various portions of the skeleton, but she still continued teaching.

Diabetes was added to her complaints in 1931.

In the Spring of 1932 she became bedridden and at that time severe pains occurred in the right and left femurs in the region of the trochanters.

In July, 1932, a complete fracture of the right femur occurred with separation of the fragments. Fracture of the left femur followed a month later.

Past Medical History. Native American, normal birth, pneumonia at two years of age, measles and mumps during childhood, acne vulgaris, for which she received many inoculations, between the ages of eighteen and thirty. She had two attacks of appendicitis but no operation. Passed a renal calculus in 1920 and 1922. Three devitalized teeth were removed in 1927. Tonsillectomy in 1927. In February, 1932, she was operated on for possible parathyroid tumor. On examination, the parathyroids appeared normal.

Family History. Father died at age of sixty-five of arteriosclerosis. Mother living and well at the age of seventy-seven. Patient is the seventh child of a family of eleven, eight of whom are living and well. One died at the age of four from pneumonia; another ten days after birth, cause unknown. Sister died at the age of thirty-eight from heart disease. There is no family history of any peculiar bone lesions, no fracture or malignancy.

Physical Examination at the Onset of Illness. Adult, female, about 5 feet, 3 inches in height; fairly well nourished; stands erect; no abnormal curvatures or bowing; does not appear to be seriously ill. She walks with a waddle gait and hesitates before starting to walk. Her weight is 129 pounds, a loss of 10 pounds in four years. On subsequent examination there was a pronounced loss of weight, with atrophy of the muscles of the extremities and chest. Her stature was markedly diminished.

Head, no deformities. Nose and throat, negative. Tonsils removed. Teeth, many filled, several missing, otherwise in good condition. Eyes, pupils round, equal, and react to light and accommodation, dark brown in color, no blue sclerotics. Neck, thyroid gland palpable; scar from operation on parathyroids. Chest, pain on pressure over sternum and ribs. Lungs and heart, normal. Abdomen, no palpable masses, no rigidity. Gynecological examinations negative for new growth or other pathology.

Skeletal Examination. Bones of skeleton are of normal growth, length, no bending, bowing or other deformities. No enlargement of epiph-

Laboratory Reports.

Blood Chemistry:

Date	Sugar	Phosphorus	Calcium	Urea N.	Uric Acid	CO_2
8- 6-26	0.094			10.5	4.6	54.1
8-12-26	0.082	(7:30 A.M.)				
8-12-26	0.093	(10 A.M.)				
10- 9-30		6.8 mg.	15 mg.			
10- 6-31	83 mg.	2 mg.?	10 mg.			
10- 7-31	111 mg.	5 mg.	10 mg.			
1-18-32	125 mg.	2 mg.	11 mg.			
1-28-32	100 mg.		10 mg.			
2-16-32	90 mg.		10 mg.			
2-15-33	(6 hour post mortem specimen taken from heart)		55.5 mg.			

Blood Count:

7-28-26: R.B.C., 4,330,000; W.B.C., 7,000; hemoglobin 81%.

10- 5-32: R.B.C., 4,430,000; W.B.C., 9,000; hemoglobin 85%; S.L., 15; L.L., 7; Trans., 1; Polys., 72; Eos., 5.

Wassermann reaction on July 30, 1926, and on repeated examinations, negative.

Basal Metabolism:

8- 5-26	+5 per cent
10-12-31	+5 per cent

Modified Glucose Tolerance Urine:

8-12-26:	7 A.M.	Vol. 465 c.c.	Sp. Gr. 1.014	Sugar 0
	12 A.M.	75 c.c.	1.020	0

Urine Analyses:

Urine examination July 28 and 30, 1926—negative. Examination from 10-3-31 to 10-12-31 shows sugar from a trace to 2 per cent and occasional acetone and diacetic acid present.

Urine examination over a period from 1-18-32 to 2-20-32 shows sugar from a trace to 5 per cent and an occasional faint trace of albumin. No Bence-Jones protein present.

Urine Calcium:

1-20-32: 24 hour specimen, 0.4 grams per day.

Feces Examination:

7-30-26: Negative.

yses, no crepitus, no abnormal mobility. Tenderness over the femurs just below the trochanters, ribs and sternum, as previously mentioned, also the right ulna. Patient is able to point out tender spots. Knee jerks ++. Blood pressure 118/78.

Roentgen-Ray Studies.

5-21-26: Examination of the dorsal and lumbar vertebrae including ribs showed no evidence of any abnormality.

7-31-26: Pyelogram negative.

8-7-26: Examination of paranasal sinuses negative.

July, 1927: Examination of the lumbar spine and pelvis was normal. In the region of the lesser trochanter of the right femur, a faint loss of cortical continuity was visualized and encircled two-thirds of the shaft. A similar process, in the same location, involved the left femur. The femur appeared to be separated into two fragments by a zone of increased radioparency and was transverse to the long axis of the bone.

For brevity, this lesion will be called "defect."

4-28-28: Examination of the skeletal system, at the Scranton State Hospital:

Skull: Bones of normal detail and outline. Sella turcica of the spherical type, not enlarged, normal.

Teeth: Nearly all present, in good condition except for many fillings.

Chest: The sixth rib on the left side, 2 inches away from the spinal articulation shows a defect 3 mm. in width. The tenth rib, 1 inch from its spinal articulation, on the right and left sides, shows a similar defect. The eleventh rib on the right and left sides, $3\frac{1}{2}$ inches from the spinal articulation, shows defects. These defects are symmetrical as to bony involvement, extent and location.

Cervical, dorsal and lumbar vertebrae are normal.

Pelvis is normal.

The right femur, in the location previously described, shows the defect extending from the inner cortex and stopping at the outer cortex. The edges of the bone above and below this defect are not sharply outlined.

A similar process is seen in the left femur, identical in location.

Clavicles, scapulae and humeri are normal.

In the right ulna, at the junction of the upper and middle thirds, there is a small circular area of calcium deficiency starting in the cortex and extending into the medulla. In the lower third of the left ulna, a similar defect, though not so advanced, is noted.

The carpals, metacarpals and phalanges show no abnormality.

The characteristic roentgen finding in the lesions described above appears to be a spontaneous loss of cortical substance. Its first roentgen appearance is a small punched out area in the cortex. Later, it is increased and extends transversely, involving the medulla and outer cortex. The resulting space seems as if the bony substance had been erased and the erased area is wavy or irregular in outline and is nonopaque or of increased radioparency. This cannot be called a fracture because it is not a sudden or violent separation of bone but rather a slow progressive absorption of bone substance without any replacement of the bone. The tissue that holds the fragments together is the ground substance of bone or decalcified tissue. It cannot be considered a pathological fracture because the bone substance is not destroyed or replaced by any primary or metastatic malignant cells or inflammatory process, nor is there any evidence of cyst formation.

The multiple involvement of the skeleton, the direction of the lesion, the identical location of the defects in many paired bones separate this disease from any known entity.

7-19-28: Examination reveals all the defects previously noted and their extent increased. The seventh rib on the right side shows a defect 3 inches away from the spinal articulation. Two small defects are noted in the left ulna. Marked narrowing of the bodies of the eighth and ninth dorsal vertebrae is noted, the intervertebral space is preserved.

12-1-28: The defects of the femurs have completely encircled the entire bone. In the right fibula, about 2 inches below the head, there is a transverse defect. One-half inch below, a smaller defect on the interosseous border is seen. There is an increase in atrophy of the skeleton.

8-22-29: Examination shows an increase in calcium absorption; the cortices merge irregularly with the spongiosa. The ninth rib on the right and left sides shows a defect in the axillary margin with angulation of the fragments. This is the first time that angulation appears. The left scapula shows a defect 1 inch below the neck in the axillary margin. There is a defect in the third metatarsal of the left foot.

10-5-30: Examination shows a generalized increase of lime salt absorption in the upper and lower extremities. There is slight bowing in the upper third of the right ulna and lower third of the left ulna. The seventh rib on the right side shows a defect in the axillary line with slight deformity. The eighth rib on the right side, $1\frac{1}{2}$ inches from the spinal articulation, shows a defect.

10-5-31: Examination shows the following new defects: Fifth and sixth ribs on the left side and the eighth rib on the right side in the axillary line. There is slight outward displacement of the lower fragment of the left femur. The upper end of the right fibula shows beginning outward rotation. Atrophy of the upper extremities is increased, especially in the carpals, metacarpals and phalanges.

2-1-32: Lungs are normal. There is no evidence of marked bowing of the entire skeleton. A defect of the acromial end of the left scapula is noted.

8-8-32: Examination shows a fracture of the right femur at the site of the defect with separation and overlapping. The left femur is nearly fractured. The second and third ribs on the right and left side near the spinal articulation show new defects. There is marked kyphosis of the dorsal vertebrae.

Post-mortem roentgen examination made on February 15, 1933, two hours after death shows no defect or atrophy of the skull. Complete fractures of the right and left femurs. The dorsal spine shows multiple crushed vertebrae from the sixth to the tenth. The lower cervical vertebrae were also crushed but films could not be made of them. There are defects in the following ribs: first, second, third, sixth, seventh, eighth and ninth on the right side, tenth rib on the right side shows defects in three places and the eleventh rib on the right side. The second, third, fourth, sixth and eleventh in one place, and the eighth, ninth and tenth in two places on the left side. The twelfth rib is not involved.

Fig. 1. Defect starting at lesser trochanter and encircling entire cortex. Symmetrical as to extent and location. No deformity of pelvis. Multiple phleboliths in bladder region. Cortex of femurs preserved. Four years after first roentgen appearance.

The lumbar spine is practically unchanged. The pelvis shows no deformity. The right fibula shows the defects as reported. The left fibula is not involved. The acromial end of the left scapula shows a defect. The neck of the scapula shows a defect and 1 inch below the neck in the axillary margin is another defect. The right scapula is not involved. The right and left ulnas show the defects as reported but more pronounced. The right and left hands show tremendous atrophy but no defects.

The following bones show no defects: the humeri, although markedly atrophied, tibiae, tarsals, carpals, metacarpals, phalanges, bones of the face and skull, lumbar vertebrae and pelvis.

There were forty-three defects noted in the entire skeleton.

Fig. 2. Multiple symmetrical defects as to location and extent involving tenth and eleventh ribs. Six years after first examination. Note slight if any deformity of ribs. No overlapping or displacement. Compression of bodies of eighth and ninth dorsal vertebrae.

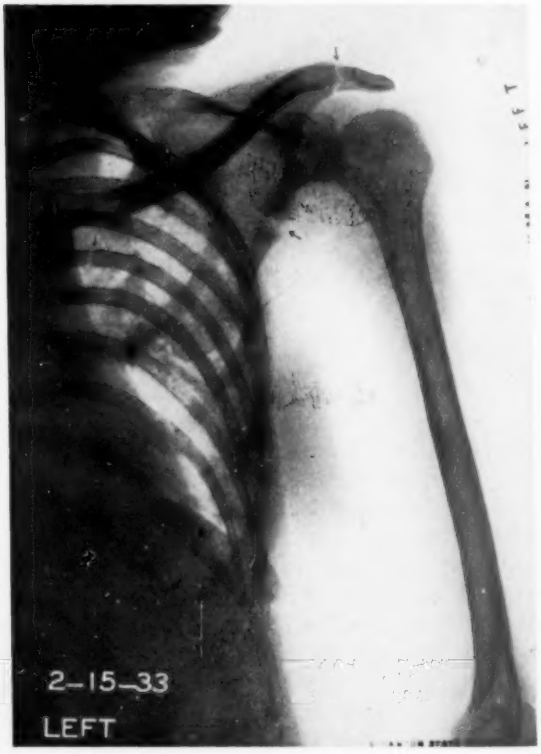

Fig. 3. Defect at acromial end of clavicle and through the axillary margin of scapula, also sixth rib. Note marked atrophy of shaft of humerus. The humeri showed no defects.

Post-Mortem Examination. The body is that of a deformed, wasted white female about forty-five years of age. The left hip is abducted and rotated inward with the left knee resting on the right knee. The body length was 4 feet 7 inches.

The following tissues were removed: brain, thyroid gland, and a careful search was made for any aberrant parathyroid tissue but none was found, parathyroid, heart, lungs, liver, spleen, pancreas, both adrenals, kidneys, ovaries, left femur, right fibula, skull and ribs.

Microscopic Diagnoses.

Brain: (1) early arteriosclerosis; (2) terminal hemorrhages, hypothalamic; (3) normal cortex. (Report by N. W. Winkelman, M.D., Temple University.)

Liver: Parenchymatous degeneration.

Spleen: Congestion and chronic splenitis.

Kidneys: Diffuse nephritis with arteriosclerosis.

Heart: Myocarditis.

Lungs: Bronchopneumonia.

Adrenals: Congestion and arteriosclerosis.

Ovaries: Congestion, arteriosclerosis; many of the vessels present hyaline degeneration.

Thyroid: Increase of colloid tissue and thyroiditis.

Parathyroid: In section of thyroid tissue, cells resembling parathyroid cells were found.

Left femur: Osteomalacia of the fragile type. (Above reports by C. L. Mattas, M.D.)

Left femur: This does not resemble osteomalacia or rickets. In my experience, this microscopic picture is constant in the bone dystrophies of the congenital type and the word osteopsathyrosis is used by us to cover brittle bone, marble bones and a group of other rare dystrophies including this. (Report by Charles F. Geschickter, M.D., Johns Hopkins Hospital.)

Fig. 4. Defect lower third of ulna showing angulation but not completely separated. No periosteal reaction. Fragments appear to be held together by ground substance.

Fig. 5. Early lesion in upper third of right ulna. The process begins as a punctate area gradually extending transversely until it involves the entire shaft.

Right fibula: The histopathological picture indicates clearly the disease osteomalacia; the age of the patient, lack of association with pregnancy and resistance to nutritional measures indicate a comparatively rare senile form. (Report by R. P. Custer, M.D.)

The difference in pathological reports is due to the fact that all atypical bone conditions are classified pathologically if they show any signs of softening, or if the pathologist finds any osteoid tissue, as osteomalacia. If fractures occur in the presence of osteoid tissue, it is also called osteomalacia by some pathologists, and fragilitas ossium by others.

Bone pathology is one of the most difficult fields in pathology. The line separat-

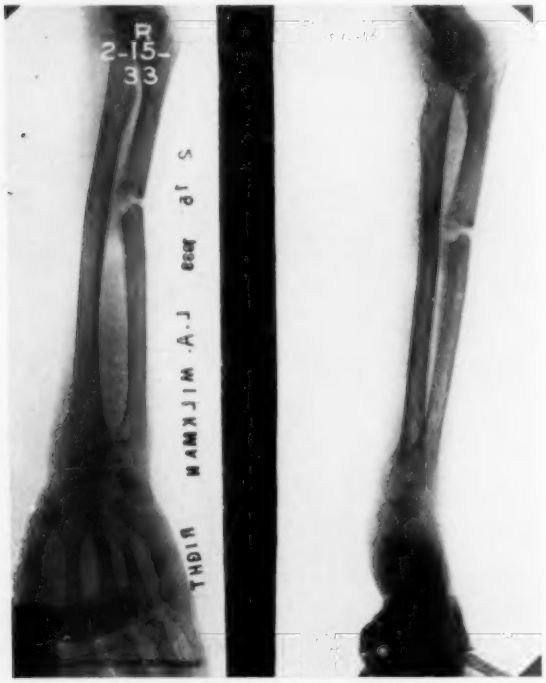

Fig. 6. Five years later. Advanced atrophy. Defect in upper third of ulna, complete, showing bowing but no displacement of fragments.

Fig. 7. Six years after first roentgen appearance. Complete fracture of right femur with overlapping and displacement of fragments. Almost complete fracture of left femur with marked angulation. Note no gross deformity of pelvis although patient was bedridden for two years. Atrophy of pelvis not marked.

ing the various bone diseases is not sharply defined and frequently overlaps, as more than one factor may be responsible for the diseased condition.

Bone physiology may be able to explain the disturbances of bone function rather than bone pathology, which is the end-result of a series of events. Bone physiology is unable to clear up the intermediate events, and until it can supply the missing data, there will be a gap between bone pathology and the clinical findings in bone diseases. More attention must be given to the study of bone physiology.

Pommer states that bone is not a static tissue but bone deposition and resorption occur during the entire lifetime; also during growth there is a slow but steady upbuilding of bone. Atrophy of bone is due to diminution or slowing in the apposition of bone rather than to an increase in absorption of bone.

In the disease described, it is not only a disturbance of deposition or an increase of the absorption of the calcium, but it is a peculiar pattern type of absorption which is atypical for any known disease entity, and it cannot be explained by any known theory. It must be due to some control mechanism in the nervous system which regulates small localized trophic changes. This control may be indirect through the blood supply of the involved parts. This seems to me to be confirmed by the pathologists' reports of increased vascularity of all the involved osseous tissue.

Of all the classified bone diseases we have either bone production, bone destruction, mass bone atrophy or combinations of one or all of the above factors. The bone production may be regular, following the normal pattern of the particular bone, or it may be irregular, deviating from the normal bone pattern as in new growth. Bone destruction, resulting from the invasion of the bone by either organisms or cells, with their secondary changes, can be recognized. Bone atrophy, whether due to lack of function, lack of nutrition, disturbance in its nerve or blood supply, or endocrine imbalance, lends itself to study and can be classified.

Disturbance of the parathyroid gland function has been proved capable of pro-

ducing osteitis fibrosa cystica, and perhaps osteitis deformans. When the above factors are further complicated by multiple glandular disturbance and dietary deficiencies, plus lack of good hygiene and sunlight, a group of diseases, which we call rickets in childhood, late rickets in adolescence, and osteomalacia at maturity, are produced.

When osteomalacia is mentioned, again there is a conflict of theories. In Kaufmann's Pathology the definition of osteomalacia is as follows: "When the term osteomalacia is used, it is usually in the sense of an unknown general disease of the skeleton, particularly in certain parts of the world such as Lombardy, Cologne, Westphalia, East Flanders, etc." In other words, he realizes the confusion and describes it as the particular kind of osteomalacia with which he is acquainted. He does not want to confuse it with other types of osteomalacia described by different authors in different localities.

Puerperal osteomalacia is another type, associated with pregnancy and lactation.

Experimental osteomalacia is produced in animals, but pathologists are not willing to agree that the changes are real osteomalacia.

A typical picture of osteomalacia is softening of the bone, marked calcium absorption, marked deformities, especially of the pelvis and femurs. The blood calcium is low, phosphorus may be normal. Fractures may occur but they usually heal with callus formation.

Osteomalacia that occurs in India, as described by Agnes C. Scott and also by Grace Stapleton, shows marked softening and deformities of the bones, especially the pelvis, and simulates the puerperal type. The fractures occur, and may be multiple, but are usually healed by a good callus weld. The hygienic conditions of the Indian women, the Purdah system, religious customs, and diet deficiency, all help to produce the more or less usual type of osteomalacia.

The above picture differs markedly from the disease described in that the blood calcium is normal, there is no deformity of the pelvis, the fractures are multiple and symmetrical, there is no attempt at repair and the post-mortem study of the bones shows no evidence of softening except at the involved parts. The sections had to be cut with a saw because they were hard. No brittleness or crumbling of the bone was noted. There was no response to medication. In the case described the patient was well nourished, did not suffer from any dietary deficiencies, and the hygienic conditions were excellent.

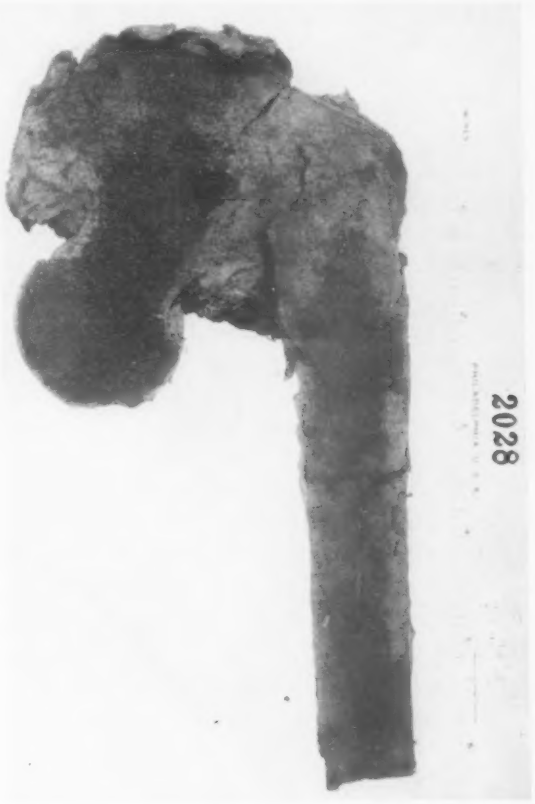

Fig. 8. Photograph of specimen section through left femur showing the irregular line of fracture and deformity, yet the relatively normal appearance of the osseous tissue.

Although there are frequent famines in India and China, there are no reports of hunger osteopathy in these regions. It may be that death occurs before bone lesions are produced. Among the survivors there is no osteopathy reported. Also, in individuals who are hunger artists, while they lose a

great deal of weight, there is no report of skeletal changes. Therefore, hunger in itself is not the main etiological factor.

In Germany, Austria and Poland during the war, especially from 1918 to 1920, there appeared a great number of patients with symptoms referable to the skeletal system, and were reported in the literature under the following titles: late rickets, osteomalacia, hunger osteopathy, pseudofractures, peculiar diseases of the skeletal system, etc. The feeling of most of the European authors, namely, Alwens, Eisler, Fromme, Looser, Porges and Wagner, and Simon and Nirenstein, was that the etiological factor was the lack of food. Yet, it is to be noted in the literature that many of the patients came from the rural districts where food was not a factor. Some of the cases described in Austria correspond to osteomalacia, especially in that the disease incidence occurred among females of an older group age. The ages were from forty to sixty years. Some of the cases corresponded to senile osteoporosis and affected many old men. In Germany, many young men were affected, and Looser considered these cases as late rickets, or the severe form of late rickets which he calls juvenile osteomalacia.

Diseases of the skeletal system assumed endemic proportions in 1919, and the public recognized it and called it "marmalade legs." They blamed it on a diet consisting largely of adulterated preserves. The lack of food seems to have been uppermost in the people's minds, and therefore everything was blamed on food shortage.

In 1921 Simon made a survey of the diseases affecting the skeletal system and reported his findings under the title, "Late Rickets and Hunger Osteopathy." His description of the symptoms follows: One of the first symptoms was pain in the legs, which soon was associated with difficulty in walking and a peculiar inelasticity of the gait. Climbing or descending stairs became exceedingly difficult. These symptoms were generally overlooked or mistakenly diagnosed as rheumatism. Accompanying this pain there was tenderness of the bones, especially at the epiphysis of the knee joints or occasionally of the vertebrae on percussion. The age incidence, from fifteen to eighteen years, was particularly singled out, and there were three or four times as many males as females involved. He attributes this to the comparatively hard work to which the young adult males were subjected. This peculiar sex distribution is not satisfactory in view of the fact that osteomalacia is most common and predominates among the females. In Vienna and Berlin there were no reports of increased puerperal osteomalacia.

In Looser's article, there are photographs and copies of roentgenograms showing fractures and transparency zones, but the deformity of the bones, the marked calcium absorption, would classify the cases as late rickets and osteomalacia. His article contains thirty-eight clinical illustrations and eighty-seven roentgenograms, and not one corresponds to multiple spontaneous idiopathic symmetrical fractures.

Wilton has again brought up the controversy regarding fetal chondrodystrophy. It goes back to Glisson, who considered it a form of fetal rickets. The rachitic origin of chondrodystrophy seems to have been disregarded in favor of such causes as endocrine imbalance, thyroid, parathyroid and pituitary anomalies, systemic disease of the mesenchyme. Wilton considered the etiological factor of chondrodystrophy the same as in rickets, vitamin D deficiency, parathyroid hormone deficiency and disturbance in mineral metabolism. He further subdivides the substances that affect the normal development of cartilage into two groups: (1) Those substances which accelerate the biological process of development, namely, vitamin D, phosphorus and parathyroid hormone, Wilton calls the K factor. (2) The antagonistic substance which retards this growth activity (P factor). For normal development the ratio K/P must have a certain value. If the ratio is too small, development is delayed, cartilage and cartilaginous bones are affected

and fetal rickets develops. At a later date, the cartilaginous and membranous bones are involved (late rickets) and in adult life connective tissue, bone and teeth are affected (osteomalacia and caries of the teeth of pregnancy).

If these factors were so from a therapeutic standpoint, it proves that the disease entity, multiple spontaneous idiopathic symmetrical fractures does not belong to the group described by Wilton for the following reasons: (1) There is no evidence of involvement of the parathyroid glands, as the blood calcium came within normal limits on repeated examinations at long intervals. (2) At operation and at post mortem there was no evidence of tumor of the parathyroids. (3) Vitamin D was supplied as phosphorized cod-liver oil and viosterol. (4) Sunlight and ultraviolet radiation were given over long periods of time until the entire body became tanned. This was repeated on various occasions. (5) There was no evidence which might suggest rickets or late rickets either on physical examination or on roentgen study.

Recently N. S. Lucas stated that it is possible to produce vitamin D by irradiation through the skin and a sufficient amount of light is transmitted through the skin to reach the ergosterol and activate it. This has been done experimentally in animals. Therefore, in the case described, the K factors of Wilton were sufficiently supplied, but due to disturbance or abnormality in the metabolism, the patient was unable to utilize artificial vitamin D, and unable to produce vitamin D in her own body. Whether there was an absence of ergosterol in her tissues, or whether it could not be activated by irradiation, is unknown. Whether the P factor of Wilton was increased there is, at present, no way of determining, except from the fact that clinically the patient showed no signs of repair of any of the multiple spontaneous symmetrical fractures. Even after the occurrence of complete separation of the fragments there was no attempt at repair, no callus formation, no bony reaction, showing that the repair factors were either absent, markedly suppressed or unable to function. Calcium was given to the patient but she was unable to utilize it. Perhaps that was an expression of the disturbance of calcium metabolism, either because the hormones were inactivated, retarded or were unable to be utilized by the tissues.

Michaelis describes a systemic disease of the skeleton in a young man, eighteen years of age, who had his onset in 1917 and is still under observation. He shows the same multiple symmetrical type of fractures as are described in this paper, with the added involvement of the bones of the pelvis and a small defect in the skull. Michaelis' patient's history, preliminary diagnosis, and later diagnosis correspond to the various diagnoses considered in the case presented. Michaelis had done various biopsies on the parathyroids, on the involved bones, and is also unable to reach a pathological diagnosis. The following pathologists examined the tissues: Doctors Schmorl, Pick and Axhausen. The histological preparation of the specimens was partly unsatisfactory due to the fact that they were completely decalcified because the diagnosis of multiple myeloma was made, and it was urgent that it be ruled out.

Summary of Histological Findings. There is absolutely no evidence for the diagnosis of myeloma, also no evidence of fracture, infraction or regeneration zones. On the nature of the disease process, the authorities have nothing definite to say.

It is remarkable that Michaelis, whose case is reported from the University of Breslau, and the pathologists, who are familiar with the various osteomalacias and hunger osteopathy seen during and following the war, did not classify his case as belonging to that particular group. Michaelis, himself, was unable to come to any definite conclusion. He suggested it might be a new disease entity, but was not certain.

The above facts help to sustain my contention that multiple spontaneous idiopathic symmetrical fractures is a definite disease entity, and does not belong to

the rachitic osteomalacic group of diseases.

Lobstein, in 1825, described idiopathic osteopsathyrosis or fragilitas ossium. Later, Gurlt, Griffith, Broca, Herbult, Looser, Axhausen, Lipschutz, Matswoka, Eckman, Vonrebbling and more recently Wagoner and Sharpe have added to the literature. According to the various authors, the disease usually starts in early childhood or adolescence. Wagoner's case is one of the oldest reported, thirty-eight years of age. The fractures occurred in early childhood and have continued during the patient's lifetime. There was marked deformity of the bones due to malunion. Fractures occur as the result of slight trauma and later are repaired, hence, are not spontaneous. In the literature, there is a hereditary factor mentioned, also blue sclerotics and difficulty of hearing.

One of the pathologists diagnosed the bone tissue as osteopsathyrosis or fragilitas ossium and considers it a form of congenital bone dystrophy. He also feels that it is due to a defect of the mesenchyme.

The disturbance of the mesenchyme is hardly tenable in the case described, because of the late onset of the disease. The patient was well until thirty-five years of age as shown by history, physical examination and roentgen examination of the osseous system made in 1925 and 1926.

Furthermore, in multiple spontaneous idiopathic symmetrical fractures there is no sudden fracture of the bones, as seen in fragilitas ossium. The bone is gradually destroyed and after a long period, complete separation and disalinement of the fragments may occur with no tendency to repair.

In the case here presented, there is no evidence of any history or stigmata usually associated with idiopathic osteopsathyrosis and differs from osteopsathyrosis in these details: (1) it is not familial; (2) no blue sclerotics; (3) no difficulty in hearing; (4) no evidence of repair; (5) the symmetry of the lesion as to location and degree; (6) lack of deformity; (7) no history of trauma. This condition differs from the type of fragilitas ossium known as osteopetrosis or marble bones, Marmorknochen, Marmorskelett, Albers-Schönberg's disease, diffuse osteosclerosis, etc., in that it shows no evidence of sclerosis of the osseous system, no epiphyseal deformities, physical underdevelopment, hydrocephalus, anemia, lymphocytosis and other characteristics usually associated with osteopetrosis.

CONCLUSIONS

1. A new skeletal disease, which is progressive, fails to respond to medication and may end fatally, is reported.

2. The case history, with resports of serial roentgen examinations over an eight year period with post-mortem findings, is reported.

3. The characteristics of this disease are the disturbance in gait, pains in the back, and peculiar multiple symmetrical involvement of the skeleton.

4. The characteristic roentgenographic appearance is bands or zones of increased transparency seen throughout the involved bones of the skeleton. They are multiple and symmetrical. Forty-three fractures occurred.

5. Differential diagnosis is made between this disease, late rickets, osteomalacia and fragilitas ossium.

6. Pathologists differing in their interpretation of the histological tissues show the limitation of bone pathology in its ability to differentiate obscure osseous changes.

7. Parathyroid glands are not involved so far as pathological study is able to determine.

8. The etiology is unknown. The postmortem findings of increased vascularity at the transparency zones suggest some trophic disturbance.

While it may be a worthy aim on the part of the pathologist to try to unite groups of bone diseases into certain distinct groups as von Recklinghausen, Looser,

Schmidt, Wilton and others, nevertheless, the scientific attitude is first to separate the different entities into definite, distinct, individual diseases before grouping under one disease entity, a variety of complicated, conflicting pictures.

I wish to express my thanks to Drs. Butzner, Propst and Noecker for their clinical coöperation.

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PRIMARY RECTAL CARCINOMA UNDER RADIATION TREATMENT

A STATISTICAL REVIEW OF 500 CASES*

By HARRY H. BOWING, M.D., *and* ROBERT E. FRICKE, M.D.
Section on Therapeutic Radiology, The Mayo Clinic
ROCHESTER, MINNESOTA

THE first important advancement in the treatment of carcinoma of the rectum was surgical, and for many years surgical intervention was the only method of treatment. With the advent of radium and roentgen rays, however, the field of treatment has been extended, and radiotherapy in this field is destined to be of as much value as surgical treatment has been; from a combination of the two methods great benefits will be inevitable.

From 1915 to 1931 at The Mayo Clinic there were more than 800 cases of carcinoma of the anus, rectum and rectosigmoid in which treatment was given with radium and roentgen rays, either alone or in combination with some surgical procedure. As a preliminary report, this paper presents the results of analysis of 500 of these cases. The study yielded considerable data from the standpoint of the clinician, surgeon, and pathologist, and it seemed of special value to the radiologist since these cases may be considered in part a selected group referred for radiological consideration and treatment.

To facilitate evaluation a number of tables were prepared. There are, however, definite limitations to this method of analysis; for example, not all records contain complete data. This is especially true of the early records when carcinoma evidently was considered wholly inoperable and, for the best interest of the patient, preliminary studies and observations were reduced to the minimum. In the recording of the rectal complaints for this study only the principal one was selected, to prevent complicated or involved statistics. The histories were analyzed as the general file furnished them and no attempt was made to select special cases for this study, except that as many cases as possible were taken from the early years of the period. From the years 1929 and 1930 six cases only were obtained in order to have representative records of the later histories for comparison. The number of cases of each year, which were analyzed, was as follows: 1915, four cases; 1916, eight; 1917, twenty-seven; 1918, twenty-four; 1919, fifty-five; 1920, seventy-three; 1921, fifty-eight; 1922, forty-eight; 1923, fifty; 1924, thirty-six; 1925, thirty-three; 1926, twenty-nine; 1927, twenty-one; 1928, twenty-eight; 1929, two, and 1930, four. The majority of the patients, 385, or 77 per cent of the entire group, were in the three decades between forty and sixty-nine years of age (Table 1). The youngest patient was a youth aged eighteen years, and the oldest a man, aged eighty-six years. The average age was 53.04 years. There were 345 males in the group, or 69 per cent, and 155 females, or 31 per cent. The average age of the males was 54.57 years and of the females, 49.63 years.

TABLE I
AGE INCIDENCE

Decade of life	Patients	Per cent	
1	0		
2	4	0.8	
3	23	4.6	
4	46	9.2	
5	106	21.2	
6	149	29.8	77
7	130	26.0	
8	39	7.8	
9	3	0.6	
10	0		
Total	500	100	

Youngest: 18 years (male)
Oldest: 86 years (male)
Average age: 53.04 years

* Read before the American Congress of Radiology, Chicago, Ill., September 25–30, 1933.

The situation of the rectal carcinoma is very important to the radiotherapist. Lesions low in the rectum are more accessible than those in the region of the rectosigmoid. This feature may influence the selection of radiation treatment, and therefore the distribution such as occurs in this study cannot be compared with that of an unselected group. In 250 cases, or 50 per cent of the group, the lesion occurred high in the rectum; in 189, or 37.8 per cent, it occurred in the ampulla, and in 61, or 12.2 per cent, it occurred in the region of the anus.

The situation of the malignant neoplasm in the rectum was further subdivided (Table II) in accordance with the opinions of the clinician, surgeon, and proctologist; in some cases, the exact situation was difficult to decide. Anatomically, the rectum extends from the level of the third sacral vertebra to the level of the lower and anterior part of the prostate gland in the male and to the upper level of the perineal body in the female. The rectosigmoid is that portion of the intestinal tract made up of the terminal 5 cm. of the sigmoid and the proximal 4 to 5 cm. of the rectum. Digital examination is a limited procedure, in that the length of the index finger, its diameter, the size of the anal canal, and other factors restrict the accuracy of estimation by this method. In many instances also, digital examination is very painful, and, therefore, limited in its application of necessity. Surgically, much importance is attached to the relation of the neoplasm in the intestine to the reflection of the pelvic peritoneum. In many instances the size or extent of the lesion made it impossible to be certain regarding the exact situation of the primary lesion.

A suitable classification of cases is indispensable. The oldest method of grouping them is from the standpoint of operability, and the judgment and skill of the surgeon, the judgment of his associates, and hospital facilities in the preoperative preparation and postoperative care of the patient enter into this method of classification. Many additional factors guide the surgeon in the selection of cases for operation which may be listed as follows: the age, sex, obesity, marked loss in weight, or general health of the patient, the duration of the disease, and the size of the tumor, its situation, and mobility, and so forth.

The method of classification employed in this study may be considered a combination, in that the clinical and surgical data as contained in the file furnished the basis for the classification as listed in Table II. Some lesions were reported as having been considered operable but if, at the time of the abdominal exploration, involvement of regional or distant lymph nodes or metastasis to the liver was found, the lesions were

TABLE II

SITUATION AND CLASSIFICATION OF RECTAL TUMORS

Situation	Cases		Classification of tumor according to operability					
			Operable		Borderline		Inoperable	
	Number	Per cent	Number	Per cent	Number	Per cent	Number	Per cent
Anus	12	2.4	6	50.0	1	8.3	5	41.6
Anus and rectum	49	9.8	19	38.7	7	14.2	23	46.9
High rectum	50	10.0	20	40.0	3	6.0	27	54.0
Low rectum	189	37.8	81	42.8	15	7.93	93	49.2
Middle rectum	120	24.0	53	44.1	9	7.5	58	48.3
Rectosigmoid	80	16.0	37	46.2	4	5.0	39	48.7
Total	500	100.0	216	43.2	39	7.8	245	49.0

Recurring: 71, or 27 per cent
Modified: 19, or 3.8 per cent

classed as inoperable. Other features entered into the classification of operability, such as attachment or fixation to pelvic structures, marked local secondary infection, and so forth. In the main, the inoperable group contained the extensive local lesions. The borderline group was made up of those lesions that approached the inoperable state. In the recurring group were placed all lesions that recurred while patients were on our own surgical service. The modified group represented those lesions that apparently had been inadequately treated elsewhere, that is, the primary lesion had been modified and seemingly favorably influenced.

It is of interest to note that the situation of the primary lesion in the rectum did not materially influence the classification according to operability. In all likelihood there is no discrepancy, especially when it it recalled that surgery in the early years was considered the method of choice in dealing with malignant rectal lesions. In 433 (86.6 per cent) of the 500 cases, some type of surgical procedure had been carried out for the primary lesion, or colostomy had been performed here or elsewhere. The notes on the surgical card greatly influenced the data contained in Table II.

The site and extent of the primary lesion, of course, influences the surgeon in the selection of cases. In this group of cases, the operable and inoperable lesions are about equally distributed among the various situations, especially if the borderline class are given a plus or minus value and are added or subtracted from either the operable or inoperable figures.

In Table III the chief rectal complaint is tabulated. When one considers that more than half of the patients in this study may be considered to have extensive or inoperable lesions, it is obvious immediately that more than one rectal complaint was recorded. To obviate a complicated compilation only one complaint was selected, as a rule the one the patient stressed most, usually the most persistent or constant complaint. Complaints referable to some degree of obstruction were almost impossible to segregate. When the patient complained of inability to have satisfactory movement of the bowels, the complaint was listed as "obstruction phenomena." However, if pain, tenesmus, "diarrhea," desire to go to stool frequently, and constipation are considered due to some degree of obstruction, the number of cases of obstruction would be very high (240, or 48 per cent); yet this would be in keeping with the other data referable to the incidence in the in-

TABLE III

CHIEF RECTAL COMPLAINT

Chief rectal complaint	Frequency		Average duration	
	Number	Per cent	Years	Months
Melena, bleeding, hemorrhage	215	43	2	6
Pain, tenesmus	90	18	2	1
Obstruction phenomena	71	14	1	10
Diarrhea, desire to stool, frequent stool	67	13	2	3
Mass in rectum	24	5	1	8
Constipation	12	3	2	8
Lack or loss of rectal control	11	2		6
Mucus, pus	7	1		6
Irritation	1		1	5
Hemorrhoids	1		11	6
None	1			
Total	500	99		

operable group. No attempt was made to determine the most common complaint with reference to the site of the primary lesion. One is tempted at the possibility of assigning certain complaints as characteristic of neoplasms in certain situations in the anus, rectum and rectosigmoid.

The average duration of the chief complaint is recorded in Table III. The average patient in this group evidently was not impressed with the importance of an adequate examination at the onset of the complaint or sign of a rectal lesion. The diagnosis, as a rule, was readily established, and was confirmed by a satisfactory history, and by digital and proctoscopic examinations. Biopsy is necessary to establish the grade of pathologic change, which is very important information to have in estimating the method of attack and the probable prognosis.

It is most important to evaluate the bodily or physical status or strength of the patient. A careful general examination, with the necessary laboratory analyses and reports, are necessary for this estimation. With this information we are in a better position to judge or to evaluate the best possible procedure to employ, in order that the best result can be obtained and the rates of surgical and radiation mortality and morbidity may be reduced to the lowest possible figure. We must always keep in mind that it is impossible to determine the true significance of these findings and impressions in a given case, so that when all factors are considered, there is still the possibility that some patients with potentially serious associated conditions will tolerate surgical and radiological intervention very well.

An attempt was made to list the many associated pathological conditions that were recorded. The 500 patients exemplified 656 conditions as follows: diabetes mellitus afflicted 4 patients; hypertension, 26; syphilis, 9; achylia gastrica, benign gastric lesions and duodenal ulcer, 4; endocarditis, myocardial degeneration, cardiac hypertrophy, tachycardia, auricular fibrillation, 25; obesity, 35; arteriosclerosis, angina pectoris, 51; renal insufficiency, 2; emaciation, marked weakness, 82; secondary anemia, 56; hepatic insufficiency, cirrhosis, 3; emphysema, chronic bronchitis, 4; carcinoma of lip, 1; carcinoma of uterine fundus, 1; carcinoma of breast, 4; benign neoplastic lesions (prostatic hypertrophy, fibroma, and myoma), 33; loss of weight, 266; *Endamoeba histolytica*, or ulcerative colitis, 3; external hemorrhoids, 19; carcinoma of larynx, 2; fistulas (rectovaginal, rectovesical, sacral), 5; gallstones, cholecystitis, 18; tuberculosis of lung and omentum, 2; Parkinson's disease, one. In 53.2 per cent of the group, a loss in weight was noted and in 16.4 per cent, emaciation and marked weakness were noted. Secondary anemia was noted in 11.2 per cent, and this, in all likelihood, is a very low figure and should not, therefore, be accepted without a more careful estimation.

Tissue studied by the pathologist was removed for biopsy or was obtained from the operative specimen, and was available in 319 (64 per cent) of the cases. The growth was papillary adenocarcinoma in 9 cases (2.8 per cent), adenocarcinoma in 249 cases (78.0 per cent), colloid carcinoma in 48 cases (15.0 per cent), squamous cell epithelioma in 11 cases (3.4 per cent), and melano-epithelioma in 2 cases (0.6 per cent). Some reports originally read adenoma; however, following a second biopsy, or if a more representative specimen was studied, a diagnosis of adenocarcinoma was made, which might be explained by the fact that adenoma tends to undergo malignant degeneration.

In Table IV the grade of pathological change is tabulated. Of the 319 lesions studied, 220, or 69 per cent, were graded according to Broders' method.

Table V is not intended to show results from treatment, the purpose is instead to point out its value as a prognostic feature. The art of foretelling the course and termination of a disease is a challenge to every physician. To know beforehand, with certain limitations, of course, the probable or

TABLE IV

INDEX OF MALIGNANCY

Pathologic report	Number graded	Graded 1	Graded 2	Graded 3	Graded 4
Papillary adenocarcinoma	6	5	1	0	0
Adenocarcinoma	170	23	92	34	21
Colloid carcinoma	34	9	20	4	1
Squamous cell epithelioma	8	1	2	3	2
Melano-epithelioma	2	0	0	0	2
Total	220*	38	115	41	26
Per cent	100	17.27	52.27	18.36	11.81
		153 (70 per cent)		67 (30 per cent)	

* In 220, or 69 per cent, of the pathological reports the grade of malignant change was recorded.

TABLE V

CLASSIFICATION OF LENGTH OF LIFE BY GRADE OF MALIGNANCY

Pathological report	Patients	Total traced		Dead, time unknown	Lived less than 1 year	Length of life in years									
		Number	Per cent			1	2	3	4	5	6	7	8	9	10+
Papillary adenocarcinoma graded 1 and 2	6	6	100		2	1			1			1			1
Papillary adenocarcinoma graded 3 and 4															
Papillary adenocarcinoma not graded	3	3	100												1
Adenocarcinoma graded 1 and 2	115	114	99.13	1	18	34	13	8	10	7	5	3	3	1	11
Adenocarcinoma graded 3 and 4	55	54	98.18	1	26	9	3	5	1	3	1				
Adenocarcinoma not graded	79	77	97.46	2	29	22	9	5	1	2			1	1	2
Colloid carcinoma graded 1 and 2	29	28	96.55	1	4	8	5	3		2	3	1			2
Colloid carcinoma graded 3 and 4	5	5	100		2	1	1					1			
Colloid carcinoma not graded	14	14	100		7	4	1								
Squamous cell epithelioma graded 1 and 2	2	2	100		1	1									
Squamous cell epithelioma graded 3 and 4	6	6	100		3	1		1	1						
Squamous cell epithelioma not graded	2	2	100				1			1					
Lymphosarcoma	1			1											
Melano-epithelioma	2	2	100		2										
None	180	161	89.44	19	88	47	8	6	2	1		1	1	1	4
Scar tissue	1	1	100												1
Total	500	475	95.0	25	182*	128	41	28	16	16	9	7	5	3	22

* Died in Hospital, 15.

eventual outcome is important, in that the attack can be planned so as to accomplish the greatest good with the least possible risk. In this table the neoplasms are graded 1 to 4. It is significant that adenocarcinomas predominate. There were 115 patients with adenocarcinoma graded 1 and 2 and of these 114, or 99.13 per cent, were traced; 95 lived one year or more, 48 lived three years or more, 30 lived five years or more, and 11 lived ten years or more.

Patients with adenocarcinoma graded 3 and 4 did not progress as well, and although they did not live as long as those in the other group, in all likelihood there is some significance in the length of their lives considering the degree of the malignancy. Fifty-four of these 55 patients, or 98.18 per cent, were traced: 26 lived less than one year, 10 lived three years or more, and none lived beyond the sixth year.

Evidently, it is possible to grade colloid carcinomas. As can be seen in Table v the number of these tumors is small, but the tendency for patients with the lower grades of malignancy to outlive those with the high grades is evident. There are too few squamous cell epitheliomas in the table for comparison with other types of tumor. Doubtless epitheliomas in the rectum, anus, and rectosigmoid behave similarly to the same type of lesions elsewhere in the body.

With recent knowledge concerning carcinoma of the rectum and improved surgical procedures for its control, many of the older surgical methods employed have been abandoned or so modified that they would meet modern requirements. This is equally true of radiation treatment; for example, formerly, in some of the cases, treatment consisted of the implantation of bare glass radon seeds, but, today, glass seeds are obsolete. Only low voltage roentgen rays were available in treatment in some of the earlier cases studied.

Most of the early lesions in the rectum, anus, or rectosigmoid were considered operable by surgeons. Today, it is possible to list recognized surgical procedures, for example: colostomy and perineal excision, abdominoperineal resection in one stage, and abdominoperineal resection in two stages; local operations, such as the Quénu-Tuttle or Harrison-Cripps, and palliative operations, such as colostomy and local cauterization.

Radiation treatment of carcinoma of the rectum is equally demanding on the resources of the radiologist. Every factor enumerated above must be evaluated in selecting the technique and the one best suited to meet the needs of the patient. The radiotherapist has a definite responsibility in the treatment of neoplastic rectal disease. There are certain cases today in which surgical intervention is unsuitable and, in a goodly number of these, treatment with radium and roentgen rays is satisfactory either for "cure" or for palliation. The risk attached to the treatment is present, but when compared to the surgical risk, it may be considered very low. This risk lies chiefly in the radium puncture technique. Surface treatments can be applied with very little or no risk.

In Table VI the cases are grouped according to the surgical procedure or treatment with radium or roentgen rays employed for the primary lesion. The order in which they are listed is not in sequence. Of the group of 500 cases, in 433, or 86.6 per cent, some type of surgical procedure was employed during the course of the disease, and in 481, or 96.2 per cent, some type of treatment with radium was given during the course of the disease.

Of the total group there were 372 cases, or 74.40 per cent, in which colostomy was performed and 128 cases, or 25.60 per cent, in which colostomy was not performed. From the standpoint of treatment by irradiation this is a very important factor. In some cases in which obstruction is marked there is no choice; in other cases colostomy may be employed as a permanent or temporary arrangement. The operation entails definite risk, and, with many of the patients, there is definite diminution in their efficiency. With this consideration

TABLE VI

CLASSIFICATION ACCORDING TO PROCEDURES EMPLOYED

Procedures at Clinic and elsewhere	Cases		Colostomy	
	Number	Per cent	Number	Per cent
Surgical, with radium	92	18.4	62	
Surgical, with radium and roentgen ray	108	21.6	82	
Surgical, with radium and surgical diathermy	1	0.2	0	
Surgical, with radium, roentgen ray, and surgical diathermy	3	0.6	2	
Surgical, with roentgen ray	10	2.0	7	
Total (61 cases without colostomy)	214		153	71.00
Radium, and roentgen ray	204	40.8	161	
Radium, roentgen ray, and surgical diathermy	1	0.2	0	
Radium, and surgical diathermy	4	0.8	2	
Radium	68	13.6	50	
Roentgen ray	9	1.8	6	
Total (67 cases without colostomy)	286		219	76.57
Total cases with colostomy			372	74.40
Total cases without colostomy			128	25.60
GRAND TOTAL	500	100.00	500	100.00

433 surgical cases (primary and colostomy): 12 deaths, or 2.77 per cent. 481 radiological cases: 3 deaths, or 0.62 per cent.

in mind, it can be truthfully stated that it is possible to treat a selected group of patients with rectal carcinoma adequately without the necessity for colostomy. With many patients today the need for colostomy is impending, but it seems good judgment to delay the procedure and to treat them by irradiation, dietary measures, mild cathartics, and low cleansing enemas when necessary and then, later on, if obstruction is imminent, to select a time suitable for performing colostomy.

The surgical mortality rate for resection of rectal carcinoma varies greatly in nineteen American cities of a population of 100,000 or more. It is estimated to be as high as 49 per cent, whereas the mortality rate attending the operation when performed by a specialist in this field is but 3 to 14 per cent. The death of 12 patients in the group treated surgically, or a mortality rate of 2.77 per cent, may not be significant in that the patients in this selected group were receiving treatment by irradiation; on the other hand, it may indicate that the surgical mortality rate among patients treated by radium is very low. However, the 3 deaths, or a mortality rate from treatment with radium of 0.62 per cent, is significant in that it points out that a definite, although small, risk is present when radium therapy is employed, and especially when interstitial treatment is given.

All patients treated with radium and roentgen rays should not be placed in one class or group when the results from such treatment are to be considered. The most outstanding feature of radiation treatment is the utmost flexibility or wide range of technique possible in outlining and applying the treatment. For example, it is possible to apply treatment with the idea of "cure," or total healing, or of getting rid of the local or visual and palpable malignant lesions. Further, it is equally possible to apply cautious treatment, with very little or no risk to the patient, with the idea of diminishing some of the distressing complaints, such as rectal discharge, bleeding, and pain. This treatment may inhibit or arrest the local lesion, and thus prevent obstruction or impending involvement of the

bladder, as well as any of the possible complications due to pelvic extension or involvement.

In order to evaluate the treatment applied, the following terms are employed: complete, limited, prophylactic, and abandoned. A complete treatment is outlined and applied to deliver an adequate and equal distribution of radiant energy throughout the local lesion, whereas a limited or cautious treatment is designed to bring about some degree of palliation. The prophylactic treatment is applied usually to the operative field following surgical removal of the involved tissue. Any one of the above treatments may be abandoned for numerous reasons, for example because of weakness, fever, hemorrhage, and so forth. Every attempt is made to keep the number of patients whose treatment was abandoned as low as possible, and thus to raise as high as possible the number for whom the treatment outlined with radium and roentgen rays is suitable. It is stimulating to plan the therapeutic attack and then to see an immediately favorable result on the local lesion with satisfied expressions from the patients as they feel better. The various types of radium treatment given are outlined in Table VII.

In Table VIII the courses of treatment with radium have been tabulated according to whether they were given before, after, or independent of operation. In this table, 19 cases are excluded in which treatment consisted of irradiation with roentgen rays alone. The results may be discussed from the standpoint of "cure" or from the standpoint of palliation. Both are important since it is logical to assume that in every case the carcinoma in the rectum was in a curable stage at some time during the course of the disease. It is impossible to be certain concerning the exact time of this phase of the disease; however, an attempt should be made to establish the probability of "cure" or palliation when the patient presents himself for consideration. The factors to be considered are too numerous and variable for presentation here.

In Tables IX, X, and XI the "three year cure" and longer cures will be presented.

TABLE VII

TYPES OF RADIUM TREATMENT

Treatment	Treatment		Radium courses of treatment					
	Number	Per cent	1	2	3	4	5	6
Complete	361	53	243	74	27	13	5	2
Limited	247	36	196	32	13	3	2	1
Prophylactic	50	7	42	5	4			
Abandoned	16	3	16					
Total	674	99	497	111	44	16	7	3

TABLE VIII

RADIATION TREATMENT IN RELATION TO OPERATION

Classification (radium)	Cases		Courses of radium (cases)					
	Number	Per cent	1	2	3	4	5	6+
Preoperative	43	6.0	42	1				
Postoperative	231	33.0	153	48	17	7	5	1
Nonoperative	420	61.0	286	81	32	13	4	4
Total	694	100.0	481*	130	49	20	9	5

* Roentgen ray alone: 19 cases.

No attempt will be made to estimate the amount of palliation that has occurred, and yet this is an important feature in the treatment of carcinoma of the rectum. In this study there were 245 (Table IX) patients in whom the lesion was classed as inoperable and palliation alone was all that could have been anticipated.

Table IX presents the results of treatment in cases classified as operable, borderline, and inoperable, and in Table XI all cases have been listed according to the procedures employed.

COMMENTS

This study is an attempt to present statistical data gathered from a review of 500 cases of carcinoma of the anus, rectum, and rectosigmoid, seen early in our experience with radium and roentgen-ray therapy at The Mayo Clinic. The surgical and radiotherapeutic techniques employed in these cases were representative of the time. Many of the surgical procedures used during the early years of this study have paved the way for better surgical procedures today. Preoperative preparation and postoperative care of patients have influenced the surgical mortality rate favorably. The technique of radium treatment used in the

TABLE IX

CLASSIFICATION OF CARCINOMA AND RESULTS OBTAINED

Classification of carcinoma	Patients				3 year "cures"		5 year "cures"		10 year "cures" and beyond		Total "cures"	
	Total	Not traced	Dead, time unknown	Traced*	Number	Per cent	Number	Per cent	Number	Per cent	Number	Per cent
Operable	216	0	1	215	82	38	53	25	15	7	150	30
Borderline	39	0	2	37	8	22	4	11	3	8	15	3
Inoperable	245	12	10	223	18	8	6	3	4	2	28	5.6
Total	500	12	13	475	108	23	63	13	22	5	193	38.6

* 95 per cent.

TABLE X

RESULTS FROM VARIOUS RADIATION TREATMENTS

Classification (radium)	Patients		3 year "cures"		5 year "cures"		10 year "cures" and beyond		Total "cures"	
	Total	Traced	Number	Per cent	Number	Per cent	Number	Per cent	Number	Per cent
Preoperative	42	42	8	19.04	7	16.66	4	9.52	19	45.23
Postoperative	153	152	20	13.16	27	17.76	11	7.24	58	38.15
Nonoperative	286	264	16	6.06	5	1.89	5	1.89	26	9.84
Total	481	458*	44	9.60	39	8.51	20	4.37	103	22.48

* 95.21 per cent.

The cases were grouped under three headings in Table X. The cases in which the primary lesion was treated with radium before operation were likewise considered "preoperative"; and those in which radiation treatment followed operation on the primary lesion were considered "postoperative"; cases in which operation was not performed on the primary lesion were classed as "nonoperative." The time between treatment with radium and operation or between operation and treatment is very important, but it will not enter into the discussion.

TABLE XI

PROCEDURES EMPLOYED AND RESULTS FROM TREATMENT OF CARCINOMA OF RECTUM

Procedures	Patients			3 year "cures"		5 year "cures"		10 year "cures" and beyond		Total "cures"	
	Total	Traced									
		Number	Per cent	Number	Per cent	Number	Per cent	Number	Per cent	Number	Per cent
Surgical with radium	87	87	100	11	12.64	17	19.54	10	11.49	38	43.67
Surgical with radium and roentgen ray	97	97	100	15	15.62	14	14.58	4	4.17	33	34.37
Surgical with roentgen ray	10	10	100			1	10.00	2	20.00	3	30.00
Surgical with radium, roentgen ray and surgical diathermy	3	3	100	1	33.33	1	33.33	1	33.33	3	100
Surgical with radium and surgical diathermy	1	1	100			1	100			1	100
Radium	77	74	96.10	4	5.71	1	1.42			5	7.14
Radium and roentgen ray	211	203	96.20	13	6.59	4	2.03	4	2.03	21	10.61
Radium, surgical diathermy	4	4	100			1	33.33			1	33.33
Radium, surgical diathermy, and roentgen ray	1	1	100					1	100	1	100
Roentgen ray	9	8	88.88	1	14.28					1	14.28
Total	500	488	97.60	45	9.47	40	8.42	22	4.63	107	22.53

early cases has also paved the way for better methods. Many of the early techniques may be considered obsolete. This is equally true of roentgen therapy. The so-called low voltage equipment has been replaced by machines with higher voltages. This accounts for the lack of detailed discussion as to the methods employed in the treatment of these early cases. There has been steady progress in this field of endeavor and, naturally, the better selection of cases and the improvement in surgical and radiological techniques as well as a deeper knowledge of the pathology of this malignant neoplastic disease should insure better results in the future. These data may furnish material for comparison in order to evaluate the extent of improvement in the initial and end-results in the succeeding years in the treatment of this dreaded disease.

This study contains many variable factors; however, every endeavor was made to present the truth as furnished by this limited number of cases seen in the comparatively recent years of our experience in therapeutic radiology at the Clinic. No apologies are necessary, but allowance must be made for the fact that this study covers a period of transition.

In the very near future the total 800 and more cases seen during this period will be presented.

CONCLUSIONS

1. Surgical intervention of cases of carcinoma of the rectum, anus, and rectosigmoid is most important and should be the first consideration.

2. Colostomy is a necessity in some cases. It should always be considered as a

means of establishing a permanent or temporary opening. It is possible to apply adequate radiation treatment in selected cases without colostomy.

3. An attempt should be made to estimate the grade of malignancy in each case and then to decide on a plan of attack.

4. Therapeutic radiology, especially radium therapy, has a distinct place in the treatment of carcinoma of the rectum, anus, and rectosigmoid.

5. Preoperative radium therapy should receive special consideration and, when employed, should be followed by a period sufficiently long, probably eight to twelve weeks, before surgical intervention is attempted.

6. Radium therapy as a palliative procedure is of value, and inoperable and recurring lesions should be given at least one well planned treatment. The degree of palliation naturally varies, but nearly all of the patients will be benefited somewhat.

7. Radium therapy as a postoperative measure has a limited field of usefulness; all lesions of a high grade of malignancy at least should be treated.

8. Roentgen therapy is of value, and with the increased voltage of the present day installations, should become of greater value, especially in cases in which lesions are of the higher grades of malignancy.

9. Since the rectal polyp may undergo carcinomatous degeneration, adequate treatment or removal of these lesions may be classed as a procedure to prevent the occurrence of carcinoma of the rectum.

10. Owing to the advanced state of malignant involvement of the bowel in the majority of the cases today as well as the age and general physical condition of the patients, and so forth, the combined efforts of surgery, therapeutic radiology, and medical treatment should greatly reduce the surgical mortality rate and enhance the initial and late result.

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RADIATION TREATMENT OF CARCINOMA OF THE CERVIX*

By WILLIAM P. HEALY, M.D.,
Attending Surgeon, Department of Gynecology, and
A. NORMAN ARNESON, M.D.,
Clinical Research Fellow
Memorial Hospital
NEW YORK CITY

THIS paper is a preliminary report of 26 cases of cancer of the cervix in which our usual plan of external irradiation with roentgen rays was changed in order to increase the amount of effective radiation given to the pelvic field adjoining the cervix, with the thought of controlling the disease in advanced cases for a longer period of time, and possibly thereby to increase the percentage of permanent cures in such cases.

Carcinoma of the cervix is now generally recognized as a malignant lesion which can be controlled reasonably well in the favorable cases by radiation therapy. In early cases in which the disease is limited to the vaginal cervix, we can expect to obtain about 50 to 60 per cent of five year cures even with radium alone and without the addition of external irradiation. However, such favorable cases form a very small group of the total number suffering from the disease.

About 75 to 80 per cent of the patients already have parametrial involvement and intrapelvic metastases as well as a greatly enlarged and often ulcerated infected cervix when they are first seen. Such cases offer an unfavorable field for application of radium and, moreover, radium applied in the region of the cervix alone cannot have a curative effect upon cancer elsewhere in the pelvis, since effective radiation cannot be expected further than 3 to 4 cm. from the cervical canal with the filtration and dosage usually used.

For these reasons since 1921 at the Memorial Hospital we have resorted routinely to roentgen irradiation for the distant pelvic involvement and radium for the cervical lesion. Since 1929 we have advocated and have given the roentgen-ray cycle before application of radium in all but the earliest cases. This has seemed to be advantageous in diminishing the discharge as well as the bleeding, and in reducing the gross size of the primary lesion by causing the superficial, infected and friable tissues to disappear. As a result, there is also less swelling and induration in the surrounding tissues. In this way, the cancer field is in much better condition to react satisfactorily to the application of radium, and usually the local and constitutional reactions are less severe.

There is nothing new in the principle of combining external roentgen irradiation for the entire pelvic field with the local application of radium to the cervix. Döderlein of Munich has carried on this method since 1913 and claims to be the first to have done so.

In this country and in some of the larger clinics in Europe, external irradiation has not been favorably received and in fact has been and still largely is omitted from the routine treatment of cancer of the cervix.

We have carried on this method of treatment at the Memorial Hospital faithfully for twelve years and while we know that we have done very much more to relieve the symptoms and prolong the lives of the large number of advanced cases seen by us than we could have accomplished by the local use of radium alone, we have nevertheless failed to increase materially the total five year salvage above that obtained by those who restrict their treatment to the local application of radium to the cervical lesion.

* Read before the American Congress of Radiology, Chicago, Ill., Sept. 25–30, 1933.

At no time have we assumed or claimed that the amount of effective radiation obtained in the deeper portions of the pelvis from one high voltage pelvic cycle delivering 700 r to the skin surface of each of four fields given within a week would be sufficient to bring about complete disappearance of cancer in the parametria. However, we are optimistic enough to believe from our observations during this time that a method can be devised by which this may be accomplished through the use of external irradiation.

This report deals with an attempt to control parametrial disease by increasing the amount of external irradiation with roentgen rays so as to obtain a greater intensity of radiation within the pelvic structures without causing severe damage to normal tissues.

The routine pelvic cycle employed at the Memorial Hospital consisted of 700 r to each of four fields with the following factors: 50 cm. target skin distance, 0.5 mm. copper and 2 mm. aluminum filtration, 200 kv. peak voltage, 30 ma., 10×15 cm. field.

In order to obtain greater intensity of effective radiation in the deep pelvic tissues it is evident that the skin dosage must be increased and, in addition, the focal distance may be lengthened, thereby increasing the percentage depth dose. To increase the skin dose above the erythema dose obtained by a single exposure it was necessary to plan a cycle of multiple exposures to each field.

In order, therefore, to accomplish this we changed our previous roentgen-ray factors by increasing the target skin distance from 50 cm. to 70 cm. and the number of roentgens delivered to each field from 700 r to 2,000 or to 2,400 r. Treatments of 200 r were given to an anterior and a posterior portal for a total of 400 r each day, alternating the right and left sides daily. These were continued until each skin area had received the total dosage planned, which required from twenty to thirty days.

When the roentgen treatments were finished, radium was applied in the vaginal canal against the cervical lesion for approximately 1,500 millicurie-hours, the filtration consisting of 2 mm. brass. Immediately following this treatment, two radon capsules were placed in the cervical and uterine canals for 3,000 mc-hr., the filtration consisting of 0.5 mm. gold and 2 mm. black rubber. This combination of roentgen rays and radium represents the completed treatment in this series of cases. Histologically there were 25 squamous epidermoid cancers and 1 adenocarcinoma. All except one of the cases were clinically advanced, the disease having extended well beyond the cervix.

When we began the work we planned to give only 2,000 r to each field but when 13 cases had been treated it was our opinion that the skin and the patient would stand a heavier dose so the remaining cases, also 13 in number, each received 2,400 r to a field. The skin reactions were of special interest and were closely observed. Each field received 200 r every forty-eight hours until the total of 2,000 to 2,400 r had been given. In 10 per cent of the cases no skin reaction could be detected. In 10 per cent there was a marked skin reaction indicated by peeling and in 80 per cent the skin reaction was moderate, equivalent to a little more than a threshold erythema. Dealing as we were with an ulcerated lesion involving an organ which can be inspected frequently and from which biopsies may be readily taken we were able to follow closely, both clinically and histologically, the progress of the cases during the treatments. In practically all of the cases there was prompt response to treatment which was quite noticeable in the first two weeks. In the second two weeks, on the other hand, the response was slower and varied considerably. The time required for complete primary healing of the cervical lesion by roentgen irradiation alone varied from three to six weeks and was more prompt, as one might expect, in the group which received 2,400 r to each field. Biopsies taken from 17 cases at intervals of three to five days during the course of treatment veri-

fied histologically the clinical evidence of regression but indicated that permanent primary healing and others, though having healed completely as far as the ulcer was

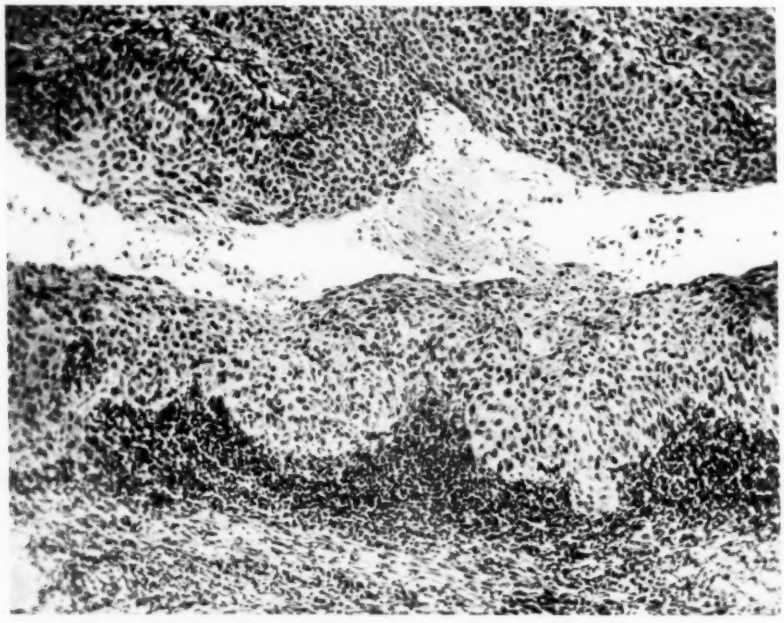

Fig. 1. C. M. Before treatment. Plexiform epidermoid carcinoma, group 2.

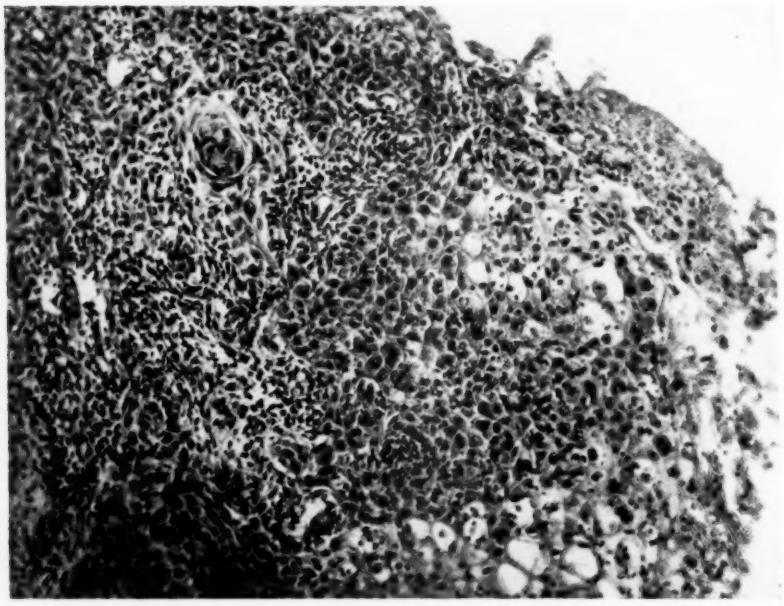

Fig. 2. C. M. First week of roentgen treatment. Plasma cell infiltration. Tumor cells swollen and becoming acidophilic; tendency toward squamous metaplasia.

healing could not be expected with this amount of external radiation alone. Some of the cases failed to show even complete concerned, still showed evidence of disease within the deeper cervical tissues which had not responded to irradiation.

Histological changes were observed within a few days after the beginning of external radiation therapy. The earliest manifestation of radiation effect consisted of plasma cell infiltration, and evidence of a diminution in any existing inflammatory process.

During the first week of treatment of the case shown in Figure 2, the cells became larger and more acidophilic, often showing a tendency toward squamous metaplasia. during the fourth week (Fig. 5). However, in biopsies taken in the weeks following, actively growing tumor was invariably found among those patients who had not yet received radium treatment (Fig. 6).

It is interesting to note that even in the presence of clinical evidence indicating a primary cure, positive biopsies were obtained (Fig. 7). The blood vessel changes persisted and fibrosis remained, but the

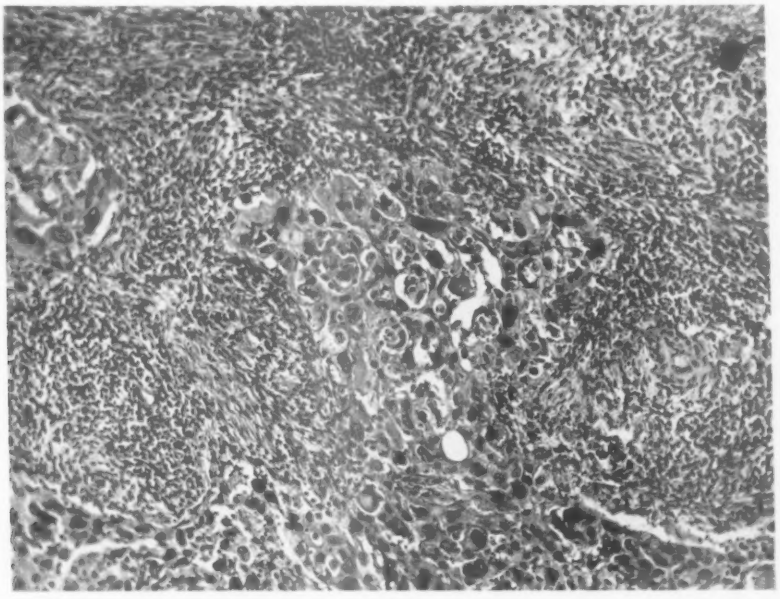

Fig. 3. C. M. Second week of roentgen treatment. Heavier infiltration of plasma cells and lymphocytes. Tumor cells more swollen and becoming hydropic; beginning arteritis and fibrosis.

In the second week (Fig. 3) the changes were more marked, the cells becoming very swollen, vacuolated, and many degenerated, hydropic nuclei could be seen. The cells developed an opaque appearance that may be said to resemble ground glass, and showed advanced hyaline degeneration. Many of the blood vessels were beginning to show evidence of arteritis and some were becoming thrombosed. The infiltration of lymphocytes was heavier, but varied considerably in different patients.

By the third week (Fig. 4) the radiation changes reached their peak. In many of the biopsy specimens very little tumor could be found, and fibrosis was more marked. It was not unusual to get negative biopsies tumor apparently adapted itself to this new environment, so that cells withstanding the effect of the roentgen irradiation were able to continue to proliferate. Ewing[1] and Frola[2] have made similar observations regarding histological changes in cancer tissues from irradiation.

As a control experiment we have also studied the histological evidence of regression in 5 patients treated at 50 cm. distance by single exposures of 700 r to each of four fields, our usual routine pelvic cycle. In this group, as would be expected, the radiation changes developed more rapidly, so that the maximum histological effect occurred in about seven to ten days. The radiation damage did not appear to be as extensive

as that in the divided dose group, so that there was less disturbance of the tumor bed. The presence of actively growing carcinoma in biopsy specimens taken two weeks after the beginning of the single exposure cycle afforded evidence that resistant cells were again multiplying. In no instance has there been complete primary healing with the cycle of 700 r to each field whereas 20 cases in the 26 here reported

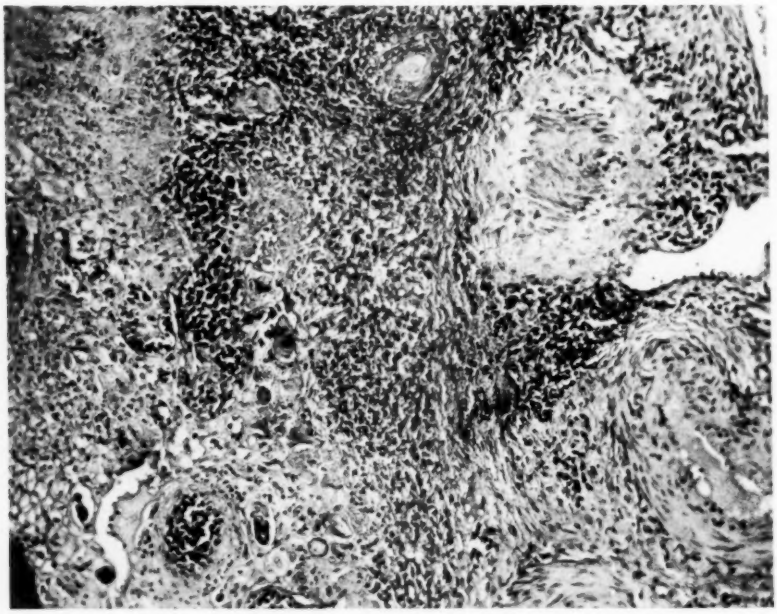

FIG. 4. C. M. Third week of roentgen treatment. Markedly hyalinized and degenerated tumor scattered in focal areas. Blood vessels thrombosed and almost completely obliterated; increased fibrosis.

FIG. 5. C. M. Fourth week. Roentgen treatment completed. Lesion epithelialized; blood vessels obliterated; marked fibrosis; lymphocytic infiltration diminished; biopsy negative for tumor; clinical evidence of primary cure.

gave primary healing in three to six weeks. The constitutional effect of roentgen cal condition, requiring them to be hospitalized throughout the entire treat-

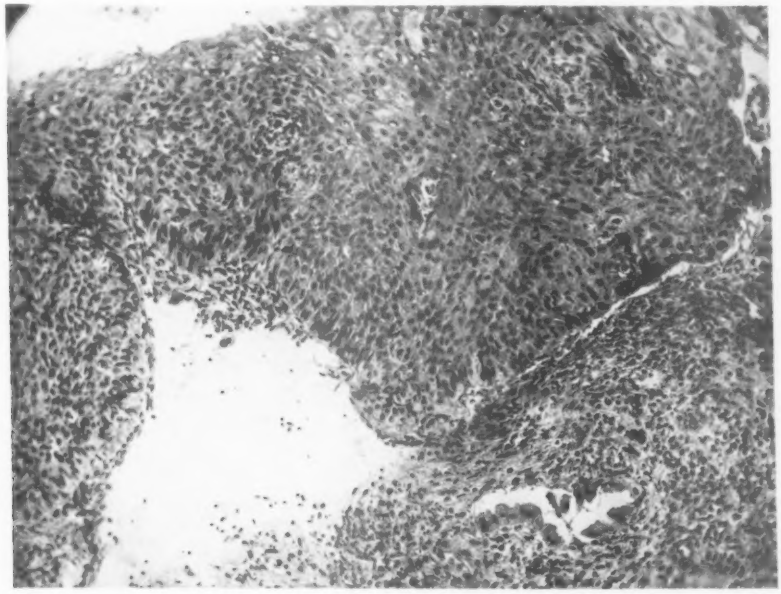

Fig. 6. C. M. Sixth week. Two weeks after completion of roentgen treatment. Biopsy obtained from cervical canal (no radium). Renewed activity of tumor. Patient still shows clinical evidence of primary cure.

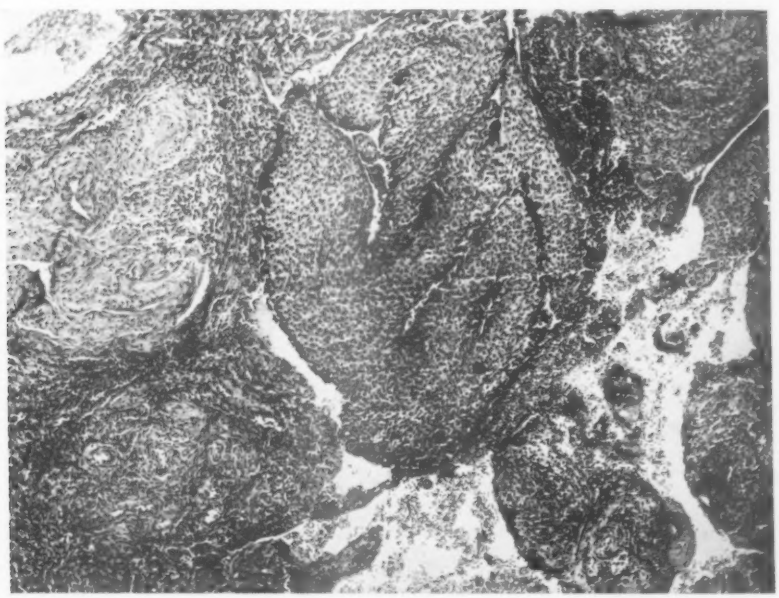

Fig. 7. A. Z. Eighth week. Four weeks after completion of roentgen irradiation. Biopsy taken at time of radium treatment. Active tumor growth. Many partially obliterated blood vessels. Clinical regression excellent. Cervix shows evidence of primary cure.

treatments given by this method was entirely satisfactory. Despite the fact that several of the patients were in poor physiment, they were nevertheless able to take daily treatments without untoward effect. This observation was very gratifying to us.

Neither were there any severe local reactions in the form of cellulitis or hemorrhage. It was our impression that the patients all stood the treatments remarkably well. There was little or no bladder or rectal distress and the skin reactions were rarely severe, seldom amounting to desquamation or peeling even toward the end of the treatment.

DISCUSSION

The dosage factors for roentgen irradiation of the pelvis described in this paper are as follows:

Voltage-peak	200 kv.
Current	30 ma.
Target-skin distance	70 cm.
Filter	0.5 mm. Cu+2.0 mm. Al
Number of skin portals	4
Size of each portal: (2 anterior and 2 posterior)	10×15 cm.
Dosage per field (each treatment)	200 r*

* Measured in air.

A plan of multiple exposures was followed so that an anterior and a posterior field of one side were treated on the same day. Daily treatments were given, alternating the right and left side until a total dosage of 2,000 or 2,400 r had been delivered to each skin field over a period of three to four weeks. From the larger treatment a tissue dosage of more than 5 threshold erythemas (Fig. 8) could be obtained in the cervical region of the average patient. The dosage delivered to tissues lateral to the cervix was less, but the bulk of the parametrial region received from about 5 threshold erythemas directly adjacent to the uterus, to 3 threshold erythema doses at a distance of 10 to 11 cm. from the cervical canal. This amount of radiation is considerably more than could be obtained in the same region by the earlier method of external irradiation with single exposures of about 700 r, which delivered approximately

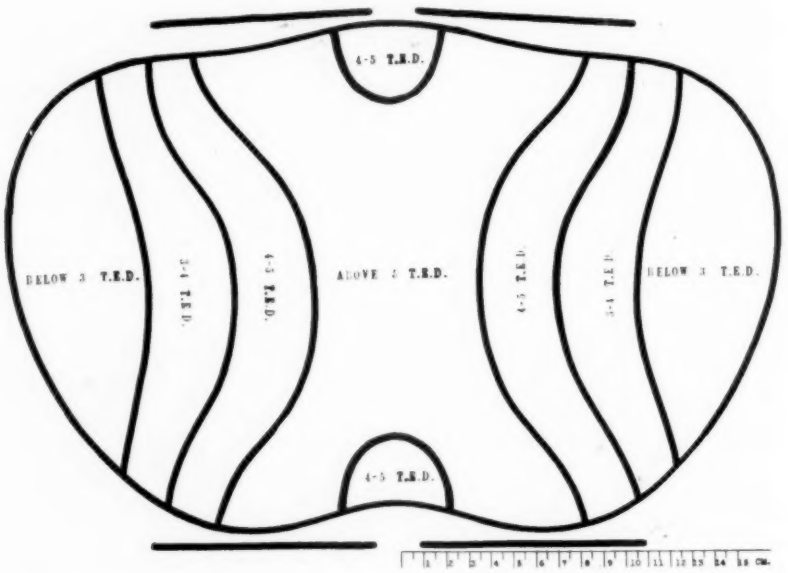

FIG. 8. Distribution of radiation in pelvis; roentgen therapy completed.

2 threshold erythema doses to the bulk of the parametria over a period of four to eight days. The application of radium added greatly to the dosage obtained in the cervix and adjacent tissues, but had little effect more than 4 cm. lateral to the cervical canal (Fig. 9).

By comparing the effects produced on the skin and in the tumor, it is evident that the diseased tissues were damaged considerably more than were the normal structures. This substantiates the selectivity of effect displayed by radiation for malignant cells.

Gunsett[3] has attempted to control the treatment of carcinoma of the cervix with external irradiation by measuring the dos-

age obtained in the cervix by means of an ionization chamber placed in the vagina at each exposure. He found that dosages less than 2 or 3 threshold erythemas were insufficient to control the disease. He also states that 3 threshold erythema doses can be delivered to the intestine over a period of five to six days without any untoward effect. In our series of 26 patients the tissue dose in the intestines was above 5 threshold erythemas delivered over twenty to thirty days, and no complications were noted.

4. We believe that the opportunity offered to observe the coincident changes in the primary lesion may be utilized to advantage to follow the effects taking place in the parametria.

5. Radium, however, should always be used for treatment of the primary lesion.

6. In the earliest cases, with a small lesion limited to the cervix and without gross enlargement of the cervix, the radium treatment should be carried out before the roentgen-ray cycle is given.

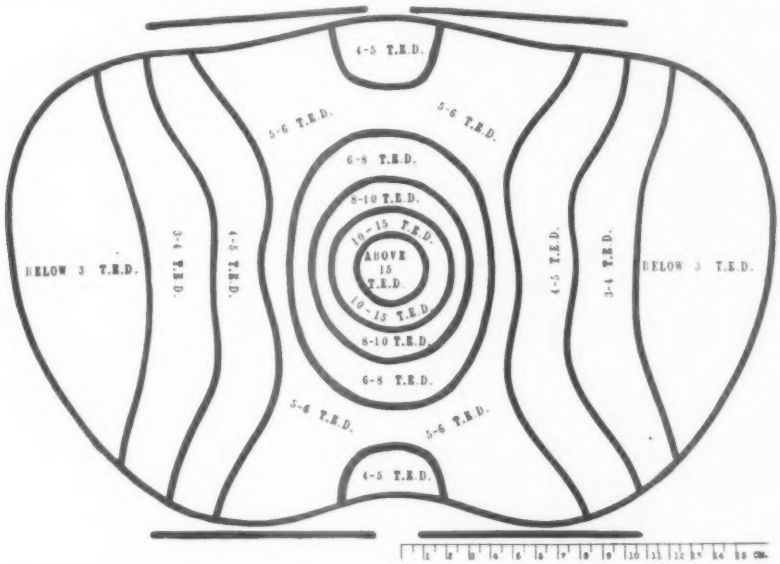

FIG. 9. Distribution of radiation in pelvis; roentgen rays and radium.

CONCLUSIONS

1. Twenty-five cases of squamous epidermoid cancer and one adenocarcinoma, all involving the cervix, were treated with roentgen rays, 200 r to each of two pelvic fields daily for a total of 2,000 to 2,400 r without severe local or constitutional reactions.

2. Clinical and histological evidence of regression of the cancer in the cervix indicated that regression might also be reasonably expected in the disease in the parametrium for this amount of external roentgen irradiation.

3. It would seem possible through these observations to plan a roentgen-ray pelvic cycle which will deliver a tissue dosage adequate for control of cancer in the parametrium and outlying pelvic field without severe damage to normal structures.

We wish to express our indebtedness to Mrs. Edith Quimby of the Biophysics Department for her cooperation and advice in checking over all the data dealing with physical measurements and determinations as well as for many valuable suggestions; to Dr. Fred Stewart of the Pathological Department who carefully checked and reviewed all the histological specimens and on whose opinion we relied in regard to the microscopic evidence of tissue changes resulting from the radiation; also to Dr. R. E. Herendeen for his cooperation in carrying out all details of roentgen therapy desired in each case.

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HEUBLEIN'S METHOD OF CONTINUOUS IRRADIATION OF THE ENTIRE BODY FOR GENERALIZED NEOPLASMS*

By LLOYD F. CRAVER, M.D., *and* WILLIAM S. MACCOMB, M.D.
Memorial Hospital
NEW YORK CITY

THE history of total irradiation of the body goes back to 1905 and 1907 when it was proposed by Dessauer and by Wetterer. Since then a few roentgenologists have tested the effect of exposure of the entire body or large sections of the body to roentgen rays in small, intermittent doses for such conditions as resistant dermatoses and leucemia. On the whole, reports have been favorable.

Arendt and Gloor, for example, report a temporary favorable result in a resistant case of myeloid leucemia, following a dose of only 3 r anteriorly and posteriorly to the entire body. On the other hand, according to Sluys, Teschendorf has found the method distinctly limited in Hodgkin's disease because of severe general reactions. Sluys himself reports that he finds teleroentgen therapy of marked value in leucemia and Hodgkin's disease, and believes it will be an important method of the future. As a rule, the method has been simply to move the roentgen tube only far enough away from the patient, usually 1.5 to 2 meters, so that the emergent beam of roentgen rays will include all or a large section of the patient's body, and to give small exposures at intervals of a few days.

The late Dr. Arthur C. Heublein conceived the idea that in order to take advantage of the varying times of mitoses in the tumor cells, total irradiation should be of low intensity, so that it might be given continuously over a comparatively long time, and that it should be as nearly homogeneous as possible in its distribution throughout the body. He had considerable experience as a practical radiologist in heavily irradiating large sections of the body. On the basis of that experience he believed that it would be possible to irradiate the entire body without great danger. His hope was that continuous, low intensity, short wave irradiation of the entire body might be carried to a sufficient dose to control tumor processes which had metastasized widely; at least those of the radiosensitive type.

In our discussion of Heublein's proposal, various theoretical and practical considerations arose. On the practical side were the favorable reports of results obtained by others by means of irradiation of the entire body in leucemia and Hodgkin's disease. Prominent among the theoretical ideas was reference to Murphy's work on mice in inducing resistance to transplanted cancer by small doses of very soft roentgen rays to the entire body. Realizing à priori that the human organism could not tolerate a dose of roentgen rays to the entire body comparable in amount to the doses required to secure regression of radioresistant cancer in local treatments, nevertheless a hope was entertained that general irradiation in small doses might stimulate in some manner a defense mechanism in man against the growth of cancer. Murphy's experiments were carried out with extremely soft unfiltered roentgen rays, the spark gap being $\frac{1}{2}$ to $\frac{7}{8}$ inch. Kok and Vorlaender, however, used a potential of 80 kv. effective, or a spark gap of 35 cm. between point and plate. They used a filter of 0.5 mm. copper.

Their report, which was supported by Caspari, showed that in the treatment of mice bearing transplanted carcinoma they obtained at least as good results as with the optimum local dose, by using only 25 per cent of this dose, or about 20 per cent

* Read before the American Congress of Radiology, Chicago, Ill., Sept. 25–30, 1933.

of the epilation dose, to the entire body.

Other theoretical considerations, based on the belief in a direct action of radiation on tumor cells or the immediate tumor bed were: (1) the advantage of a nearly uniform distribution of the rays throughout the body in treating generalized neoplasms: (2) the possibility that great protraction of treatment would make possible the irradiation of all the tumor cells during their period of mitosis when they are most radiosensitive, with (3) the assumption that despite the protraction of treatment the intensity of radiation affecting any given cell would nevertheless remain sufficient to sterilize it. As these theories were discussed in Heublein's paper they need not be gone into more fully here.

A decision having been reached to test Heublein's proposed method, a cooperative arrangement was made so that in May, 1931, the Heublein ward, capable of continuous irradiation of 4 patients, was established in a special building at the hospital.

In December, 1931, Heublein read a preliminary report, covering the first five months of operation of the unit, at the meeting of the Radiological Society of North America. The present writers, of whom the senior was in active supervision of this work from the beginning, presented a second report in April, 1933, at the meeting of the New York State Medical Society.

The unit consists of a four bed ward, behind one wall of which is mounted a Coolidge tube operating at 185 kv. and 3 ma., placed near the ceiling so that roentgen rays emanating from it may reach the patients in all four beds without obstruction other than the filter. The two near beds are 5.4 meters, the two far beds 7.3 meters from the target. The filter, at first 5 mm. of copper, was soon reduced to 2 mm. The intensity of radiation reaching the near beds is 1.7 r per hour; that reaching the far beds is 0.9 r per hour, measured in air. The intensity can of course be varied by increasing or diminishing the thickness of the filter. The voltage, 185 kv., and the current, 3 ma., were regarded as optimum for satisfactory continuous operation of the tube, but could be varied also.

With men patients we are able to average twenty hours of exposure a day; with women, somewhat less. Taking 750 r measured in air as the clinical skin erythema dose for a single exposure to high voltage roentgen rays, the time required to deliver a "30 per cent" dose or 225 r to a patient in a far bed is 250 hours. At the rate of twenty hours of exposure a day, such a dose requires the patient to remain in the far bed for 12.5 days. To assure a more nearly uniform distribution of the rays throughout the body, the beds are turned end for end twice a day.

The control panel is made so nearly automatic that it is not necessary to have a roentgen technician to operate the unit, a trained nurse being adequate. She is at no time exposed to the rays, as she is well shielded by the lead lining of the ward and its door, and the opening of the door into the ward automatically stops the operation of the tube, as well as ringing a warning bell. In starting the apparatus the nurse has merely to throw a switch, the step-up in voltage and milliamperage being carried out automatically. An electric clock starts and stops with the operation of the tube, so that the record of the time of exposure is not open to error. An ionization chamber permanently mounted under the tube gives at the control panel a continuous graphic record of the output of the tube.

In addition to the ward, there is a room directly beneath the tube, fitted with a treatment table, so that intermittent prolonged treatments may be given at a distance three times that commonly used in roentgen therapy, namely 150 cm. It is thus possible to treat 5 patients simultaneously with one tube. The 150 cm. distance is found particularly useful in the treatment of such conditions as bulky masses of retroperitoneal lymph nodes in leucemia or lymphosarcoma. It permits a maximum

TABLE I

CASES TREATED BY INTERMITTENT IRRADIATION AT 150 CM.*

Disease	Number of Patients	Minimum and Maximum Total Dose per Patient in r (Measured in air. Usually 2 portals)	Number of Days within Which Cycle Was Given	Duration of Life after Treatment in Months at Time of Report (August 1, 1933)	Number Surviving over 6 Months Following Treatment	Number of Cases in Which No Other Type of Irradiation Was Used in This Area
Carcinoma of stomach	11	586–2400	7–39	0.1 to 8	2 (1 living)	9
Carcinoma of bladder	2	1863 and 5033	15–38	2		2
Carcinoma of lung	1	2187	15	3		1
Carcinoma of submaxillary gland	1	2400	19	10	1	1
Carcinoma of sigmoid	1	2400	21	10	1	1
Carcinoma of breast	1	2090	18	8	1 (living)	0
Carcinoma of pancreas	1	840	11	4		0
Teratoma testis	4	1300–2774	13–22	3 to 28	1 (living)	0
Hypernephroma	1	1700	15	4		0
Osteogenic sarcoma	1	4000	34	2		1
Endothelial myeloma	1	3182	27	6	1	0
Hodgkin's disease	2	612 and 2900	12–31	5 and 7	1	0
Lymphosarcoma	5	290–2200	10–21	0.7 to 12	3 (all living)	0
Myeloid leucemia	5	1000–2400	5–29	1 to 6	3 (2 living)	0
Lymphatic leucemia	6	300–1200	6–14	2 to 15	4 (2 living)	0
Lymphatic pseudoleucemia	2	979 and 1000	6–9	7 and 7	2 (1 living)	0

* As the purpose of this paper is to report the results obtained by Heublein's method of continuous irradiation of the entire body, only the above summary is given of the cases treated by intermittent roentgen doses administered at 150 cm. The filter is equivalent to 1 mm. of copper. The intensity being only 35 to 40 r per hour, this method was chosen only for those cases in which a large field required treatment, and in which we were contented with highly fractionated dosage over a long period. Its chief field of usefulness was in the treatment of bulky radiosensitive tumors, such as retroperitoneal masses in leucemia or lymphosarcoma, the spleen in myeloid leucemia, and abdominal metastases of teratoma testis. It seemed to palliate some cases of inoperable carcinoma of the stomach.

portal of circular shape 27 cm. in diameter.

Table 1 and footnote summarize briefly the cases treated at 150 cm.

It may be of interest to mention that one tube rendered service for over fourteen months, having been run for 7,482 hours.

When Dr. Heublein proposed days or even weeks of practically continuous irradiation of the entire body, possibly involving unknown and unpredictable effects upon the entire hematopoietic, endocrine and other systems, there was naturally considerable discussion before it was decided to try the experiment. As there was no experience anywhere that we knew of to draw upon, except animal experiments and total irradiation of humans by methods that were really not comparable, we felt it obligatory to proceed most cautiously. Accordingly, we began with a dose of only 5 per cent S.E.D. (37 r) for the first patients, and increased the dose step by step, attempting to keep within safe limits, and at the same time to learn the tolerance dose and the effect of this new procedure upon the subjects and upon their diseases. In the first few months elaborate tests were made on each patient before, during and following the treatment, consisting of blood calcium, phosphorus, sugar, non-protein nitrogen, CO_2, pH, icterus index, fragility test and basal metabolism, in addition to blood

cell and platelet counts. As no significant changes were found in any of the blood chemistry determinations (Table II), they were discontinued, but it was found quite essential to watch the blood cell and platelet counts.

The selection of cases was controlled by a special committee on experimental irradiation. Only patients showing widespread metastases or having general systemic involvement were accepted. Patients bearing metastatic radioresistant tumors, although not promising for palliation by this method, were chosen, provided they seemed to be in sufficiently good condition to offer a satisfactory period of observation after treatment. Since they were utterly hopeless otherwise, it seemed justifiable to try this experiment on them. Moreover, they would lend themselves better to a determination of the tolerance dose than would most of the patients with radiosensitive tumors, who might be limited to small doses. Cases of generalized radiosensitive tumors were accepted, even though they might seem suitable for the ordinary methods of palliative irradiation.

We willingly accepted a good many who had received local irradiation previously, if their condition seemed to warrant a trial of this method. Nor have we hesitated to use local irradiation subsequently or even during the course of general irradiation when it seemed unfair to the patient to withhold the obvious benefits of local therapy. Naturally, it has been more difficult to appraise the value of Heublein's method of therapy in those cases that received local treatments as well. However, we preferred to rely upon a more gradual accumulation of experience than to restrict the treatment of all the selected cases to the method of general irradiation.

Unfortunately, in May, 1933, because of economic conditions, and the expense to the hospital of maintaining the unit, it became necessary to suspend its operation. We hope to be able to resume the work soon, because we are convinced that it represents a distinct contribution to radiation therapy, and that it is a method of such value in certain diseases that it should be pursued further.

RESULTS

From May 25, 1931, to May 23, 1933 (Table III), 134 patients were treated. Thirty-five of these patients were treated twice, 9 three times, and 2 the fourth time. Local irradiation had been given previously to 85, and was used during or after the Heublein treatment in 55. The Heublein method was the only form of irradiation used in 37 cases (Table IV). The experience gained during these two years in treating 134 patients forms the basis of fairly definite conclusions as to the value of prolonged short wave irradiation of the entire body in certain generalized processes. Beginning cautiously with only 5 per cent S.E.D. over a period of two to three weeks (this extremely low intensity having been obtained by high filtration), it was found possible to increase the intensity and the dose step by step, keeping ever in mind the nature of the disease and its response to usual forms of radiotherapy. Thus, in treating leucemia we were particularly cautious, but in other diseases we eventually worked up to doses of 50 to 60 per cent S.E.D. (375 and 450 r). No higher dose has been given to the entire body of any patient but in one patient with Hodgkin's disease we gave 75 per cent S.E.D. (560 r) to the neck and thorax, shielding the rest of the body. It seems possible that some patients may be found who would tolerate somewhat more than 60 per cent S.E.D., but we would judge that few or no patients could stand as much as 100 per cent S.E.D. (750 r) over the entire body in one treatment period of three weeks.

Radiation sickness, so common with local intensive treatments, is practically absent, and there is no evidence of erythema or alopecia. In women we observed no consistent menstrual changes, but it should be noted that most of the women who were treated had passed the menopause or had amenorrhea as a result of their disease or previous treatment.

TABLE II

CHANGES IN CHEMICAL CONSTITUENTS OF BLOOD FOLLOWING TOTAL IRRADIATION

Name	Dosage r (measured in air)	Sugar mg/100 c.c.			Urea Nitrogen			Calcium			Phosphorus			pH			CO_2—volume %		
		Before	After	Change	Before	After	Change	Before	After	Change	Before	After	Change	Before	After	Change	Before	After	Change
Lymphatic Leucemia																			
G.A.	187	79	83	+ 4	14.9	14.6	− .3	13.5	11.5	−2.0	2.4	3.1	+ .7	7.43	7.41	−.02	—	—	—
E.C.	187	83	85	+ 2	15.4	14.2	− 1.2	11.3	11.0	− .3	4.1	4.6	+ .5	7.49	7.44	−.05	57	56	− 1
E.J.	187	103	114	+11	18.2	17.6	− .6	12.0	11.0	−1.0	4.0	4.3	+ .3	7.37	7.39	+.02	59	63	+ 4
W.H.	225	110	160	+50	19.8	22.0	+ 2.2	11.5	11.0	− .5	5.4	5.0	− .4	7.41	7.39	−.02	56	48	− 8
L.S.	44	87	110	+23	17.1	16.5	− .6	10.5	10.9	+ .4	4.0	4.3	+ .3	—	—	—	—	—	—
J.U.	112	103	108	+ 5	18.6	20.7	+ 2.1	12.6	10.5	−2.1	4.0	4.9	+ .9	7.59	7.48	−.11	65	65	0
J.U.	187	105	123	+18	20.4	16.9	− 3.5	13.5	11.0	−2.5	4.8	3.7	−1.1	7.44	7.41	−.03	—	—	—
Myelogenous Leucemia																			
E.J.	112	133	153	+20	9.6	6.3	− 3.3	11.5	13.0	+1.5	4.5	4.3	− .2	7.41	7.39	−.02	66	57	− 9
Pseudoleucemia																			
M.F.	180	110	105	− 5	19.6	14.2	− 5.4	11.9	13.0	+1.1	2.8	3.8	+1.0	7.40	7.39	−.01	56	59	+ 3
R.H.	187	106	123	+17	13.2	17.1	+ 3.9	12.0	10.5	−1.5	3.2	4.0	+ .8	7.49	7.41	−.08	55	55	0
R.H.	187	89	83	− 6	17.6	24.9	+ 7.3	12.1	13.1	+1.0	3.9	5.2	+1.3	7.38	7.41	+.03	—	—	—
R.H.	375	105	105	0	17.1	14.5	− 2.6	11.5	12.3	+ .8	—	—	—	7.42	7.43	+.01	49	60	+11
R.W.	65	87	105	+18	20.1	20.6	+ .5	10.6	12.4	+1.8	3.5	4.6	+1.1	—	—	—	—	—	—
Lympho-epithelioma																			
W.M.	225	159	100	−59	21.7	34.1	+12.4	—	—	—	3.5	3.4	− .1	7.37	7.35	−.02	57	48	− 9
Lympho-adenoma																			
A.C.	262	103	116	+13	11.4	12.6	+ 1.2	11.0	11.7	+ .7	4.3	4.5	+ .2	7.36	7.42	+.06	48	59	+11
Lymphosarcoma																			
H.R.	75	83	95	+12	18.4	22.2	+ 3.8	11.6	11.1	− .5	4.0	4.6	+ .	—	—	—	—	—	—
Hodgkin's Disease																			
J.B.	116	95	125	+30	13.8	17.2	+ 3.4	11.0	10.5	− .5	5.3	5.6	+ .3	7.55	7.59	+.04	—	—	—
P.B.	112	112	91	−21	14.0	13.0	− 1.0	10.8	10.5	− .3	4.4	3.7	− .7	7.59	7.43	−.16	—	—	—
P.C.	225	123	100	−23	14.5	13.2	− 1.3	12.5	11.5	−1.0	3.4	4.1	+ .7	7.40	7.42	+.02	63	69	+ 6
T.G.	225	105	105	0	14.7	12.9	− 1.8	11.5	11.0	− .5	3.9	4.0	+ .1	7.37	7.34	−.03	62	69	+ 7
J.G.	300	103	123	+20	14.5	18.2	+ 3.7	—	—	—	5.1	5.2	+ .1	7.54	7.47	−.07	—	—	—
J.G.	225	210	149	−61	13.2	15.8	+ 2.6	11.7	13.5	+1.8	3.5	5.0	+1.5	7.41	7.42	+.01	55	55	0
M.H.	225	105	105	0	13.1	10.7	− 2.4	12.0	15.5	+3.5	3.8	5.9	+2.1	7.37	7.39	+.02	60	64	+ 4
A.L.	225	149	111	−38	12.1	12.0	− .1	11.0	11.3	+ .3	2.6	3.5	+ .9	7.40	7.39	−.01	59	66	+ 7
A.S.	225	116	131	+15	8.6	5.6	− 3.0	10.5	9.0	−1.5	3.0	3.1	+ .1	7.38	7.44	+.06	56	53	− 3

TABLE II (*Cont.*)

Name	Dosage r (measured in air)	Sugar mg./100 c.c.			Urea Nitrogen			Calcium			Phosphorus			pH			CO_2—volume %		
		Before	After	Change	Before	After	Change	Before	After	Change	Before	After	Change	Before	After	Change	Before	After	Change
Endothelial Myeloma																			
E.H.	225	117	117	0	8.5	8.7	+ .2	14.0	13.0	−1.0	4.7	5.5	+ .8	7.47	7.33	−.14	58	65	+ 7
Multiple Myeloma																			
F.B.	300	82	89	+ 7	10.3	8.4	− 1.9	11.2	11.7	+ .5	5.2	4.5	− .7	7.45	7.42	−.03	57	55	− 2
Melanoma																			
R.B.	225	103	98	− 5	16.4	23.5	+ 7.1	10.4	11.8	+1.4	4.0	4.7	+ .7	7.44	7.43	−.01	—	—	—
P.K.	300	83	76	− 7	15.2	14.0	− 1.2	—	—	—	2.7	3.6	+ .9	7.40	7.42	+.02	—	—	—
W.S.	300	74	89	+15	18.2	19.6	+ 1.4	11.5	11.7	+ .2	2.6	4.0	+1.4	7.38	7.35	−.03	—	—	—
Metastatic Carcinoma in Bone (Primary Unknown)																			
C.S.	450	103	149	+46	12.1	10.4	− 1.7	12.5	13.0	+ .5	5.1	4.4	− .7	—	—	—	—	—	—
Carcinoma of the Tonsil																			
E.S.	277	117	113	− 4	15.4	20.4	+ 5.0	14.0	12.3	−1.7	3.5	3.5	0	7.35	7.40	+.05	55	52	− 3
Carcinoma of the Breast																			
R.B.	112	108	82	−26	9.7	7.1	− 2.6	11.5	10.5	−1.0	4.1	4.0	− .1	7.44	7.43	−.01	—	—	—
M.C.	112	99	103	+ 4	17.3	15.4	− 1.9	12.3	12.4	+ .1	5.0	4.9	− .1	7.51	7.43	−.08	—	—	—
A.E.	225	125	117	− 8	5.9	10.0	+ 4.1	12.0	11.0	−1.0	3.3	3.1	− .2	7.42	7.35	−.07	56	52	− 4
B.G.	112	102	103	+ 1	10.8	17.1	+ 6.3	10.7	11.0	+ .3	4.1	4.6	+ .5	7.42	7.46	+.04	—	—	—
M.M.	300	114	114	0	17.0	13.8	− 3.2	12.5	11.9	− .6	3.9	4.4	+ .5	7.40	7.37	−.03	60	58	− 2
I.N.	375	111	125	+14	18.5	18.0	− .5	12.5	12.5	0	5.0	4.8	− .2	7.37	7.34	−.03	61	64	+ 3
M.O.	112	108	120	+12	14.0	9.3	− 4.7	11.5	11.5	0	4.8	3.5	−1.3	7.50	7.45	−.05	—	—	—
E.R.	112	118	99	−19	10.3	18.5	+ 8.2	10.5	10.8	+ .3	4.1	4.4	+ .3	7.42	7.46	+.04	—	—	—
L.S.	375	117	80	−37	11.8	13.7	+ 1.9	11.5	12.0	+ .5	5.0	4.2	− .8	7.39	7.39	0	60	56	− 4
Carcinoma of the Ovary																			
S.M.	60	83	84	+ 1	7.0	7.9	+ .9	12.2	13.8	+1.6	5.2	5.0	− .2	—	—	—	—	—	—
Carcinoma of the Kidney																			
W.B.	45	91	92	+ 1	11.8	11.4	− .4	9.8	10.5	+ .7	4.3	4.6	+ .3	—	—	—	64	66	+ 2

We express our appreciation to Miss Helen Q. Woodard who made the chemical analyses shown in this table.

TABLE III

SUMMARY OF RESULTS OF ALL CASES TREATED BY CONTINUOUS IRRADIATION

Total Number of Cases	Radio-sensitive Group	Radio-resistant Group	Total
	109	25	134
Showed improvement. Still living. Time from Heublein treatment to Aug. 1, 1933	49 3 to 24 mo.	1 25 mo.	50
Showed improvement but died later. Survival period following Heublein treatment	17 2 to 24 mo.	4 1 to 8.5 mo.	21
Unimproved, but still living. Time from Heublein treatment to Aug. 1, 1933	6 3 to 12 mo.	1 2.5 mo.	7
Unimproved, died. Survival period following Heublein treatment	37 1 day to 10 mo.	19 3 wk. to 12 mo.	56

Leucopenia follows the treatment rather routinely, though apparently to no greater a degree than following massive local therapy. In 54 cases the white cell count fell below 5,000 following treatment and in 19 cases it fell to 2,000 or less, the lowest count obtained in any case being 1,000, found four months following treatment in a case of Hodgkin's disease. It should be noted, however, that in some cases mild leucopenia existed before the Heublein irradiation was given. This may be accounted for in some instances by previous irradiation, in others, by the disease itself, while in still other individuals, counts of less than 5,000 are found for which no reason can be assigned. Anemia appears in some cases.

When necessary, such effects are combatted by large transfusions, and in some cases sodium nucleotide has seemed valuable in overcoming the fall in leucocytes.

Radioresistant Group. (Table III.) In this group were 25 patients, of whom 23 died. Four of those who died seemed to have slight temporary palliation.

As an example of the radioresistant group may be taken the 12 cases of mammary carcinoma, receiving doses of from 15 to 50 per cent S.E.D. (112 to 375 r). All 12 patients died, only 3 showing slight temporary palliation. Another patient with extensive metastases in the skeletal system from prostatic carcinoma had a little temporary improvement following a dose of 60 per cent S.E.D. (450 r).

One patient with ovarian psammocarcinoma (Case 1), proved by biopsy of an inguinal node, showed improvement by this method alone, and is still living, two years and one month following the first of four treatments. In this case there was a poorly outlined mass in the left lower quadrant of the abdomen, a bulky mass in the lower lobe of the right lung, and an enlarged hard left supraclavicular node. The four treatments were given at intervals of one year, six months, and four months. The doses were 60, 120, 112, and 120 r, respectively. Following the first treatment the patient improved, but after four months was lost track of, and did not return until a year after the first treatment. When she returned she had considerable cough and hemoptysis. Following the second treatment these symptoms lessened. At the end of two years' observation of this patient we found that while there was no marked decrease in the size of the pulmonary or supraclavicular tumors, there was, on the other hand, evidence of growth restraint, as indicated by the absence of evidence of other metastases, and perhaps some evidence of regression, as the abdominal mass could no longer be palpated.

Radiosensitive Group. (Table V.) In this group were 109 patients, of whom 54 are still living. All but 6 of the living patients showed definite improvement as a result of general irradiation and have survived from three to twenty-four months since

TABLE IV-A

RESULTS IN CASES TREATED BY HEUBLEIN METHOD ALONE—RADIOSENSITIVE GROUP

	Patient	Age	Condition	Dose		Results	Duration since 1st Total Irradiation	Duration since Symptoms Began	Remarks
				Days	r Measured in Air				
Hodgkin's disease	May B.	68	Poor	8	38	Unimproved died	3 days	4 mo.	Circulatory collapse, should not have been treated
	Joseph B.	39	Poor	14	116	Unimproved died	23 days	4 mo.	Extensive disease pleural effusion
	Anna Sch.	19	Fair	14	187	Improved died later	4.5 mo.	16 mo.	Disease generalized; rbc 3,500,000 before treatment
				9 (2½ mo. later)	225				
Lymphosarcoma	Herbert Rahd.	60	Poor	7	75	Unimproved died	3 wk.	2 yr. 1 mo.	Disease generalized
Lymphadenoma	Julius Wa.	75	Poor	9	112	Unimproved (suicide)	3.5 mo.	4.5 mo.	Disease generalized
Lymphatic pseudoleucemia	Richard H.	40	Good	8	40		9 mo.	21 mo.	
			Poor	29 (7 mo. later)	375	Improved died later			
	Jacob S.	50	Fair	13	187	Unimproved died	3 wk.	2 mo.	Extensive disease
	Robert Wh.	54	Good	8	65	Improved died later	9 mo.	27 mo.	
				8 (3 mo. later)	122				
Lymphatic leucemia	Cornelius F.	66	Fair	5	112	Unimproved died	1 mo.	4 mo.	
	Leopold G.	71	Fair	5	112	Unimproved died	2 mo.	10 mo.	
	William H.	37	Fair	9	225	Unimproved died	13 days	12 yr.	Acute fibrinopurulent streptococcal pleuritis
	Oscar N.	45	Good	8	112	Improved living	17.5 mo.	21 mo.	
				9 (6 mo. later)	112				
				4 (6 mo. later)	75				
	Joseph Tav.	38	Fair	7	112	Improved (lost)	3 mo. (lost)	5 mo. (lost)	Returned to Spain
	James V.	46	Fair	11	187	Improved living	9.5 mo.	11 mo.	Marked general adenopathy; very large spleen
				3 (2 mo. later)	82				
				5 (5 mo. later)	75				
	Augusta W.	55	Fair	3	112	Improved living	6.5 mo.	14.5 mo.	
				4 (4 mo. later)	75				
Myeloid leucemia	Edward J.	35	Good	9	112	Improved living	16.5 mo.	19 mo.	
				5 (2 mo. later)	112				
				9 (5 mo. later)	150				
				7 (6 mo. later)	112				
Lymphoepithelioma	Wilhelmina Mart.	42	Poor	20	225	Unimproved died	1 wk.	7 mo.	Generalized adenopathy; primary undetermined at autopsy
Multiple myeloma	David Fitz.	63	Fair to Poor	13	225	Improved died later	4 mo.	10.5 mo.	
	Charles And.	57	Poor	3	75	Unimproved died later	1 day (died of pneumonia during treatment)	22 mo.	
	Patrick Mc.	39	Fair	13	225	Improved (lost)	23 days (lost)	7 mo. (lost)	
Wilm's tumor of kidney	William G.	5	Poor	7 (lower extremity shielded last 3 days)	101	Unimproved died	4 da.	7 mo.	

TABLE IV-B

RESULTS IN CASES TREATED BY HEUBLEIN METHOD ALONE—RADIORESISTANT GROUP

Disease	Number of Patients	Results	Time Following First Treatment
Metastatic epidermoid carcinoma	1	Died without improvement?—(Lost track of in terminal stage)	?
Psammocarcinoma of ovary	1	*Improved* living	25 mo.
Carcinoma of prostate	1	Improved slightly, died later	8.5 mo.
Metastatic adenocarcinoma primary unknown	1	Died without improvement	5 mo.
Carcinoma of tonsil	1	Died without improvement	1 wk.
Carcinoma of testis	1	Died without improvement	1 day
Melanoma	2	Died without improvement	2 mo. 2 mo
Carcinoma of renal pelvis	1	Died without improvement	1 mo.
Carcinoma of breast	7	5 died without improvement	1, 2.5, 3, 8 and 12 mo.
		2 improved slightly, died later	5 mo. 5 mo.
	16	12 died without improvement. 3 improved slightly, died later 1 improved, living.	

beginning treatment. Thirty-two of those who showed improvement are living over six months, and 8 over one year. Even of those who succumbed later, 17 showed palliation. Thus 60 per cent of the patients in the radiosensitive group showed appreciable improvement and 44 per cent were improved and are still living, while in the radioresistant group (Table III) only 5 patients out of 25 showed any improvement, and only one patient had notable improvement and still survives.

The lack of results in the radioresistant group led us to give up almost entirely further attempts to treat this group during the last ten months of operation of the Heublein unit. That is the main reason we have not been able to work up to a higher dose than 60 per cent S.E.D. (450 r) to the entire body, as most of the patients with radiosensitive tumors were treated with the smaller doses.

Chronic lymphatic leucemia (Tables V and VI) responded very well in a number of cases, and seemed to do better than myeloid leucemia, which in our experience is the reverse of the results obtained in treating these two forms of leucemia with local irradiation. Twenty-seven patients with chronic lymphatic leucemia were treated with doses of from 8 to 30 per cent S.E.D. (64 to 225 r). Of these 12 improved and were living on August 1, 1933, from five months to one year and nine months following treatment (see Case II). Three others were improved, but succumbed later. Our experience in this disease taught us that by this method no patient with leucemia should be given over 15 per cent S.E.D. (112 r) in one treatment period lasting six or seven days, and perhaps it would be better to use even less.

In lymphatic psuedoleucemia (Table V) 6 patients out of 9 showed definite regression and systemic improvement, 2 of these being still alive (see Case III).

There were 5 patients with myeloid leucemia (Tables V and VII), of whom one improved and is still living, sixteen months following the first treatment. This patient (see Case IV) was admitted with an enlarged spleen extending 13 cm. below the rib margin, and a white cell count of 36,400. He received four treatments at intervals of

TABLE V

SUMMARY OF RESULTS IN RADIOSENSITIVE GROUP TREATED BY CONTINUOUS IRRADIATION

	Hodgkin's Disease	Lymphosarcoma	Lymphatic Pseudoleucemia	Lymphatic Leucemia	Myeloid Leucemia	Multiple Myeloma	Endothelial Myeloma	Mycosis Fungoides	Lymphadenoma (Brill-Symmers Disease)	Transitional Cell Carcinoma	Lymphoepithelioma	Wilm's Tumor of Kidney	Total
Total number of patients	44	9	9	27	5	6	2	1	3	1	1	1	109
Showed improvement. Still living.	26	5	2	12	1	1?	0	1	1	0	0	0	49
Time from Heublein treatment to Aug. 1, 1933	3 to 24 mo. (2 over 1 yr.) (14 over 6 mo.)	5 to 27 mo. (1 over 1 yr.) (3 over 6 mo.)	4.5 to 10 mo.	5 to 21 mo. (3 over 1 yr.) (11 over 6 mo.)	16 mo.	(lost track of after 23 d.)		5 mo.	12 mo.				
Showed improvement but died later	4	0	4	3	3	3	0	0	0	0	0	0	17
Survival period following Heublein treatment	5 to 26 mo.		7 to 24 mo.	10.5 to 24 mo.	3.5 to 6.5 mo.	2 to 8.5 mo.							
Unimproved but still living	4	0	0	2	0	0	0	0	0	0	0	0	6
Time from Heublein treatment to Aug. 1, 1933	3.5 to 9.5 mo.			3 to 12 mo.									
Unimproved, died.	10	4	3	10	1	2	2	0	2	1?	1	1	37
Survival period following Heublein treatment	3 days to 6 mo.		3 wk. to 5 mo.	13 days to 2 mo.	2 mo.	1 day to 3 mo.	6 and 10 mo.		1 and 3.5 mo.	lost track of	1 wk.	4 days	

TABLE VI

EFFECT ON WHITE BLOOD COUNT IN LYMPHATIC LEUCEMIA

Name	Dosage in r (measured in air)	White Blood Count *Before Treatment*	White Blood Count *After Treatment*
J.A.	187	4,200	2,800
O.A.	187	86,400	59,200
	90	153,000	84,400
	75	134,400	121,600
G.A.	187	54,400	11,600 (6 da.)
H.B.	63	10,400	14,100 (3 mo.)
E.C.	187	5,000	4,700
	112	3,000	2,400
A.E	75	96,400	92,800
A.F.	112	35,200	30,000
C.F.	112	32,000	96,000
L.G.	112	73,600	67,200
W.H.	225	9,200	6,600
E.J.	187	46,000	15,600
	112	16,800	9,400
S.K.	187	11,900	7,100
M.M.	142	70,400	246,000
	112	56,400	50,400
D.M.	82	19,800	10,400
S.M.	112	4,000	3,400
	112	4,400	3,800
V.M.	112	3,600	3,200
O.N.	112	21,500	14,200
	112	8,800	13,800
B.O.	112	123,200	25,000
M.S.	187	11,300	3,400
W.S.	112	20,600	13,200
	112	87,200	37,200
L.S.	44	13,400	12,200
J.T.	112	342,400	13,500
J.U.	112	18,000	6,200
	187	116,400	9,600
J.V.	187	275,000	39,500
	82	38,400	Not taken
	75	57,600	27,200
A.W.	112	195,200	144,000
	75	201,600	202,600
E.W.	112	56,000	41,000
	112	156,800	96,000

two months, five months, and six months, the doses being 112, 112, 150 and 112 r, respectively. Following the first treatment the spleen did not diminish in size; in fact, it enlarged slowly. This finding is in accord with our experience in other diseases, that bulky tumor masses may not recede satisfactorily following use of general irradiation. The white cell count, however, dropped to 15,600 and during the fourteen months that he was under observation he was able to carry on his work as a garage mechanic, with interruptions only for his residence in the Heublein ward. Three others showed definite improvement, but

TABLE VII

EFFECT ON WHITE BLOOD COUNT IN MYELOID LEUCEMIA

Name	Dosage in r (measured in air)	White Blood Count *Before Treatment*	White Blood Count *After Treatment*
F.D.	112	22,400	24,000
	120	201,600	60,800
E.J.	112	32,800	15,600
	112	32,400	29,200
	150	36,500	32,000
	112	60,800	37,600
M.L.	112	57,600	70,400

died later of the disease, three and a half, five and a half, and six and a half months, respectively, following treatment. Only 1 of the 5 died without improvement.

Undoubtedly our failure in some cases of leucemia was due to overdosage. In other failures the disease was in too late a stage. We believe that it should be possible to secure much better results in selected cases with more experience in choosing the proper dose. Tables VI and VII show the effect on the white cell count in the cases of lymphatic and myeloid leucemia.

One patient with mycosis fungoides had had a severe generalized erythematous desquamating rash for fifteen years and had suffered marked pruritus for one year. He was given 15 per cent S.E.D. (112 r). Within twenty-four hours of his admission to the unit, the itching disappeared, and the rash had faded appreciably by the end of a few days of treatment.

Hodgkin's disease (Table V) furnished the largest single group of patients, numbering 44, for the reason that to many of this group was purposely applied a departure from our usual plan of selection of cases. We deliberately treated a number of patients who had already been benefited by local irradiation, and who showed no marked evidence of disease activity, giving them a small dose (10 per cent S.E.D.—75 r) requiring a stay of three to five days, depending on the distance from the tube. The reason for doing this was that we hoped to benefit latent lesions in bone marrow, spleen, etc. We had not done this to more than a few patients (see Cases V and VI) before we became so impressed by the well-marked and persistent tonic effects of this treatment that it became almost, and we believe should be, a routine procedure in Hodgkin's disease. We believe that in Hodgkin's disease one should not attempt to bring about regression of bulky localized masses by large doses of general irradiation; in fact, as this disease is usually more radioresistant than leucemia or lymphosarcoma, it will be found, as a rule, that the patient is unable to tolerate a general dose sufficient to reduce the local masses. These masses may be satisfactorily reduced by properly chosen doses of local irradiation, and then, we believe, the patient will receive distinct benefit by treatment of the entire body. While the rest in bed for a few days undoubtedly contributes to the tonic effect observed, yet it has certainly seemed too pronounced and lasting to be explained solely on that basis.

Of the 44 patients with Hodgkin's disease, we are thus able to report 26 who were improved and are still living—14 over six months and 2 over one year—and 4 who were improved, but died later. Fourteen were unimproved.

In lymphosarcoma (Table V), 5 out of 9 patients were improved and are still living.

Multiple myeloma (Table V), gave some encouraging results. Of 6 patients, 1 improved but could be followed for only two months, and 3 were improved but died later. One of the latter (see Case VII) showed a rather spectacular improvement. She was markedly crippled, being barely able to get out of bed without assistance. She received 50 per cent S.E.D. (375 r) in one of the near beds. Only one dose of local roentgen treatment was given, to a small area over the dorsal spine, one of the chief sources of her pain. Within two months the change in the patient's condition was remarkable. She made long motor trips and walked about alone almost normally. Unfortunately, a relapse occurred six months later, and she did not respond to a second treatment, dying about five weeks afterward.

In numerous instances in these radiosensitive diseases, such as lymphatic leucemia and pseudoleucemia, the patients show improvement in general health and beginning regression of tumor masses within a few days after they begin treatment. While rest in bed is a factor in their improvement, yet the course as a whole during and after the Heublein therapy indicates that the irradiation itself is mainly responsible for the improvement. Such

observations, together with the absence of radiation sickness or erythema, and the fact that so much of the disease is being treated, which by ordinary methods would be left untreated, lead to the suggestion that for some of these tumor processes such therapy approaches the ideal.

Certain difficulties must be recorded:

1. Since the procedure is new, lack of experience makes it difficult to decide what dose to give and how rapidly to give it. We feel certain that we overtreated some patients, particularly the leucemics. It is our impression that they do better in the far beds, that is, at a distance of 7.4 meters, and that for them it might be advantageous to restore the filter originally used, 5 mm. of copper, so as to lessen the intensity and prolong the treatment time.

2. The general irradiation does not produce satisfactory regression of bulky lesions, even the more radiosensitive ones, but it is evident that local and general irradiation may complement each other in smaller doses than would be necessary if either were used alone.

3. Leucopenia, sometimes associated with considerable lassitude, and no doubt lowering of resistance to infection, and anemia, may follow frequently, just as is the case with massive local irradiation.

4. Especially in leucemia there may be a tendency to thombocytopenia and occasionally we have seen the appearance of a mild hemorrhagic tendency.

5. With our arrangement the method was expensive to the hospital, especially since so few patients, because of the present economic conditions, could pay their board. The cost of electricity per patient is of course small, as 5 patients can be treated simultaneously by one tube. As our unit is separate from the rest of the hospital, three special nurses are required to cover the twenty-four hour period. If such a unit were constructed adjacent to a regular ward, or to the roentgen department, the running of the tube is so nearly completely automatic that there would seem to be no reason why the unit could not be operated by the nurses attached to the regular ward or roentgen department, thus lessening the cost of up-keep.

In regard to the possibility, suggested by Murphy's work with mice, of demonstrating the stimulation of a defense mechanism against cancer in man, we believe, after surveying our two years of work in retrospect, that this theory was not fairly tested, and we feel that it might be worth while, in seemingly hopeless cases of cancer, to give this theory a real trial. To do so would entail the treatment of a large number of cases of metastatic cancer, and also a number of cases which would normally be expected to develop metastases within a reasonably short period. Such cases should be treated by very small doses, and probably by much softer roentgen rays than we used. We considered taking up this problem several times, but visualizing a large mortality in the first type of case, and the uncertainties of judgment of results in the second type, we did not seriously attempt it.

Some modifications of the method may be proposed for further investigation:

1. As suggested by Palmieri, continuous irradiation might be used for a single section of the body, such as the abdomen in cases of abdominal metastases of teratoma testis, or for more than one section, the rest of the body being shielded. It should then be possible to increase the dose considerably above that which can be tolerated when the entire body is treated. In this fashion we treated one case of Hodgkin's disease with 75 per cent S.E.D. (560 r) delivered to the neck and thorax, with distinct improvement and regression of nodes.

In selected cases it might be justifiable to experiment by treating lesions in part of the body by continuous therapy, leaving the rest shielded from the direct rays.

2. The use of the method in small dosage for its effect on latent foci of disease, as in Hodgkin's disease, has already been referred to, and evidently merits further investigation.

3. For certain diseases manifested chiefly

on the surface, such as mycosis fungoides, leucemia cutis, and perhaps many dermatoses, it might be possible to secure better results by unfiltered or very lightly filtered roentgen rays, perhaps generated at a lower voltage, in such a unit as Heublein's, where the tube is at a long distance, the intensity is low, the exposure time prolonged, and the entire body surface is uniformly irradiated.

The question may well be raised as to whether the results shown here could not be duplicated, or even bettered, by the usual methods of local irradiation. Judged solely on the basis of figures, that objection is probably valid. Yet other considerations must enter into an evaluation of this work. In the first place, it must be remembered that the cases treated were, in the great majority, much more advanced than the average of cases treated by local irradiation. In fact, many were so advanced that in retrospect it appears obvious that they should not have been accepted for any form of treatment.

Another point is that as the procedure was new the early doses were entirely empirical and the first few months were largely taken up in trying to establish what the immediate effects were and what doses the patients could tolerate, so that undoubtedly in a number of cases the doses were inadequate, and, on the other hand, in some cases were too large. Moreover, this report, carrying results up to August 1, 1933, follows by only a little over two months the last treatments given. Because of the interruption of operation of the Heublein unit, necessitated by economic reasons, and the fact that the care of patients required the continuation of their treatment by other methods, it seemed best to make this report at this time, while impressions were fresh in mind and before too many complications of other treatments, etc., entered in to confuse the interpretation of results.

In dealing with such a miscellany of cases and with diseases such as leucemia, in which the clinical course varies greatly in different cases, we feel that statistics are of much less importance than the impressions formed by close observation of individual cases by workers familiar with results obtained in large numbers of cases of the same diseases treated by the usual methods of irradiation. It is for this reason that we feel less attention should be paid to our figures and that the emphasis should be placed on the impressions we have gained, illustrated by the appended case reports.

REPORT OF CASES

CASE 1. *Psammocarcinoma of ovary; metastases to left inguinal and left supraclavicular lymph nodes, and to right lung.* Sarah M., aged forty-six, housewife, admitted to clinic on May 25, 1931. History of operation in Poland nine years previously, for removal of a pelvic tumor. Present symptoms of two years' duration; dyspnea, cough, and watery expectoration, slight discomfort in chest and some fever. Examination disclosed an enlarged node or nodes, forming a mass 1.5 cm. in diameter in the sentinel node position in the left supraclavicular fossa, a questionable retroperitoneal mass, a questionable tumor mass to left of uterus, and a large mass occupying the base of the right lung. Biopsy of a right inguinal node removed at another hospital and forwarded to us showed ovarian psammocarcinoma, grade 1.

Treatment and Course. First total irradiation June 11 to June 23, 1931, 232 hours of actual exposure, dose 61 r at 7.3 meters. (Dose small, as this case was one of the first treated.)

A few weeks following this treatment general condition was improved, but there was no obvious change in the supraclavicular adenopathy or in the metastatic mass in the right lung. There was less suggestion of a retroperitoneal mass and no definite pelvic tumor could be felt. Cough had increased.

Four months after treatment cough was persistent, and the supraclavicular nodes were larger, but the patient's general condition had remained remarkably good.

After an absence of eight months, patient returned, in remarkably good condition. Cough persisted, with rusty sputum. Cervical adenopathy unchanged. No evidence of abdominal or pelvic tumor. Chest film unchanged.

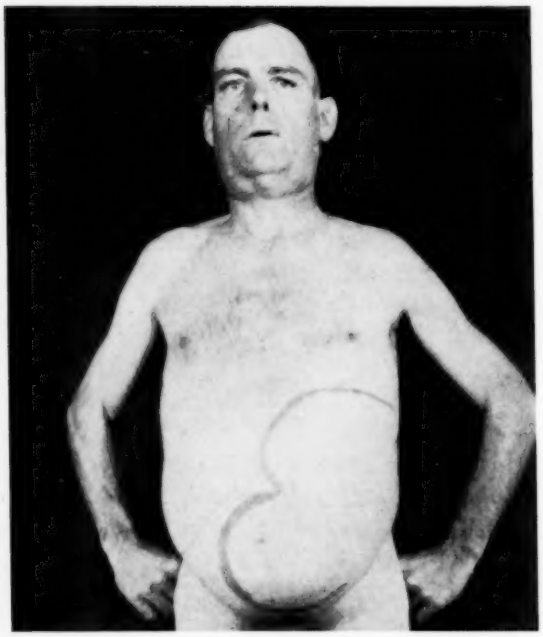

FIG. 1. Case II. Chronic lymphatic leucemia. October 7, 1932, before treatment. Edge of liver and spleen outlined.

Second total irradiation July 20 to July 25, 1932, ninety-five hours of actual exposure, dose 120 r at 5.4 meters.

After an absence of over five months, she returned, cough and bloody sputum having disappeared, but having recurred shortly before her return. Supraclavicular adenopathy less. Patient had gained weight. Chest film showed no change. Abdomen and pelvis negative.

Third total irradiation January 18 to January 24, 1933, 125 hours of actual exposure, dose 112 r at 7.3 meters.

Two months later, improvement in general condition, and decrease in cough and expectoration. No change in chest film.

Four months after the third treatment, hemoptysis recurred.

Fourth total irradiation May 20 to May 23, 1933, 59.5 hours of actual exposure, dose 120 r at 5.4 meters.

After an absence of two months patient returned July 19, 1933. General condition not as good as before. Frequent hemoptysis.

As the Heublein unit had been closed for economic reasons, local irradiation was given to the metastatic mass in the base of the right lung. Only half of the prescribed treatments were taken, amounting to 600 r posteriorly and 300 r anteriorly (200 kv.; 50 cm. distance, 30 ma.; filter 0.5 mm. Cu).

Later Note. November 14, 1933. Patient feeling very well. No cough. No hemoptysis. No change in supraclavicular adenopathy. No definite pelvic or abdominal tumor.

Comment. A metastatic ovarian psammocarcinoma of slow growth, yet apparently growth restraint and possibly some regression were obtained by rather small doses of general irradiation.

CASE II. *Chronic lymphatic leucemia* (Figs. 1 and 2). James V., male, iron-moulder, aged forty-six. Admitted to Heublein ward October 7, 1932. Family history irrelevant. Past history, pleurisy and pneumonia two years previously. Present illness: Symptoms began six weeks before admission; pain in left lower lumbar region, discovery of enlarged cervical nodes and greatly enlarged spleen; loss of 20 pounds in weight. Examination on admission disclosed pronounced general adenopathy, many intra-abdominal nodes being recognizable. The spleen

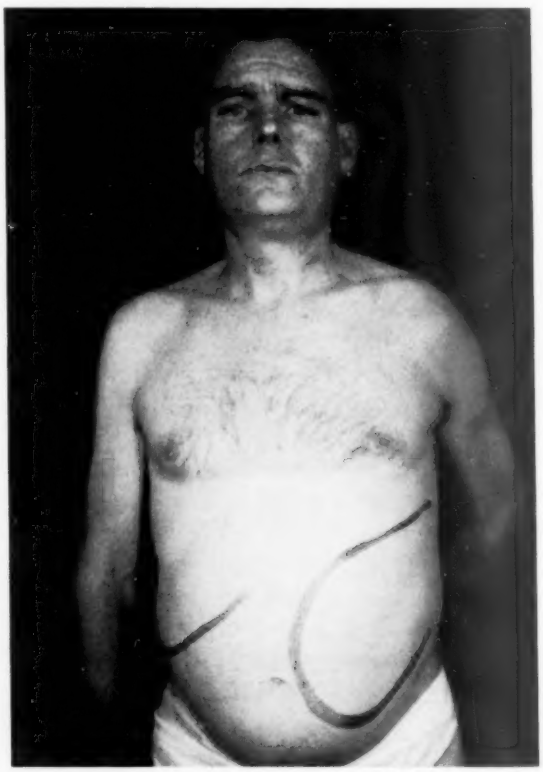

FIG. 2. Case II. December 28, 1932, ten days following completion of second period of total irradiation.

was exceptionally large, its right edge 5 cm. to the right of the midline, its lower edge being 20 cm. below the costal margin in the left midclavicular line. Blood count: Hb, 60 per cent; red blood cells, 2,976,000; white blood cells, 275,000; small lymphocytes, 100 per cent.

Treatment and Progress. First treatment. Total irradiation October 7 to October 18, 1932; 182 hours of actual exposure, dose 187 r at 7.3 meters. Regression of nodes began during patient's stay in the unit. On the tenth day of treatment the white blood cells showed a decrease to 39,500, the red blood cells having risen to 3,428,000. Three weeks later the spleen had receded so that its lower edge was 16 cm. below the costal margin in the left midclavicular line.

Second treatment. Total irradiation December 15 to December 18, 1932, forty-nine hours of actual exposure, dose 75 r at 5.4 meters. Fowler's solution 3 to 12 minims t.i.d. in ascending and descending doses. Lymphadenopathy regressed so markedly that ten days later the superficial nodes were barely palpable. General health improved and patient gained 7 pounds. One month later, January 18, 1933, the white blood cells had fallen to 21,600, with 3 per cent polymorphonuclear cells and 97 per cent small lymphocytes. The spleen was smaller.

Third treatment. The spleen and lymph nodes having enlarged and the white blood cells having risen to 57,600, total irradiation was again applied from May 12 to May 17, 1933, for ninety-nine hours of actual exposure, dose 75 r at 7.3 meters. Three weeks later the white blood cells had fallen to 12,700 with 10 per cent polymorphonuclear cells, 1 per cent large lymphocytes, 88 per cent small lymphocytes and 1 per cent eosinophiles. One month following this treatment there was only a shotty adenopathy, although the spleen remained large, extending just to the midline and 6 cm. below the umbilical level. There was comparatively little change thereafter up to the date of this report, August 1, 1933.

Later Note. The patient returned to part-time work on August 30, 1933. In October, 1933, local irradiation was used over the spleen, the Heublein unit having been closed for economic reasons. This produced a fall of white blood cells to 4,200, November 15, 1933, but no marked change in the size of the spleen. The patient has continued to work and his general condition remains good.

Fig. 3. Case III. Lymphatic pseudoleucemia. November 11, 1931, before treatment.

Case III. *Lymphatic pseudoleucemia.* Roy H., aged fifty-three, machinist, admitted to clinic, November 11, 1931. History of epigastric discomfort, vasomotor instability and weakness for ten years; bilateral inguinal adenopathy for three years. Bilateral swelling in preauricular, temporal, and submaxillary regions for seven months, generalized adenopathy for a few months, and recent exophthalmos and ectropion with impairment of vision. Loss of 25 pounds in a few months.

Physical Examination (Fig. 3). Patient underweight and pale. Bilateral exophthalmos, ectropion of right lower lid, with chemosis of lower bulbar and palpebral conjunctiva. Pronounced general lymphadenopathy; broad mound-like mass in each temple; huge mass, nodular and fixed, occupying whole central part of abdomen, extending from epigastrium to suprapubic region and across almost the entire width of the abdomen. Spleen not palpable. Chest film showed widening of the mediastinal shadow and evidence of enlarged lymph nodes at the hila. Two biopsies of lymph nodes showed features of leucemic or aleucemic lymphadenosis. Blood count: Hb, 75 per cent; red blood cells, 3,800,000; white blood cells, 6,000; polymorphonuclears, 43 per cent; large lymphocytes, 35 per cent; small lymphocytes 18 per cent; transitionals, 4 per cent; occasional fragile cells. Basal metabolic rate, plus 75 per cent.

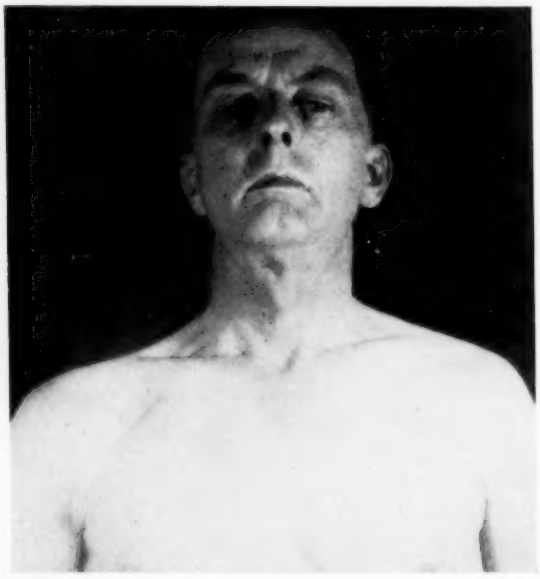

Fig. 4. Case III. December 9, 1931, eighteen days following completion of first total irradiation.

Treatment and Course. First total irradiation, November 13 to November 21, 1931, 149 hours of actual exposure, dose 187 r at 5.4 meters. At end of treatment general condition was improved (Fig. 4), lymph nodes had decreased in size, temporal and parotid swellings had gone down, and exophthalmos and chemosis of conjunctiva had entirely disappeared. Chest film showed a decrease in the size of the mediastinal mass. One month later marked general improvement in condition, and further decrease in lymphadenopathy.

Second total irradiation, January 18 to February 1, 1932, 278 hours of actual exposure, dose 187 r at 7.3 meters. This treatment was followed by further decrease in size of the abdominal mass.

Local intermittent irradiation of the abdominal mass April 16 to April 25, 1932, at a distance of 150 cm., using the table provided for that purpose directly beneath the tube (185 kv., 3 ma., filter equivalent to 1 mm. copper). One to 2.5 hours daily exposure of the abdominal mass anteriorly and posteriorly, using a rectangular portal 18×18 cm. Total dose 500 r anteriorly, 500 r posteriorly (measured in air). Further regression of the mass followed this treatment.

Third total irradiation, because of recurring general adenopathy and beginning exophthalmos, and in the face of leucopenia (2,000 W.B.C.): June 1 to June 9, 1932, 149 hours of actual exposure, dose 187 r at 5.4 meters. Intramuscular sodium nucleotide injections, 10 c.c. daily for seven days, at the end of which time the white cell count had risen to 5,100. At end of irradiation period white cell count was 8,300. One month later, marked general improvement. Patient had returned to work, but two months following treatment symptoms had recurred, accompanied by evidence of an early destructive lesion in the upper end of the right humerus, and infiltration of the base of the right lung. Local irradiation was attempted but was discontinued because of leucopenia (1,800 W.B.C.) and the poor condition of the patient.

Patient died November 11, 1932. Autopsy at Bristol Hospital, Connecticut, was restricted to the abdomen. "Except for a marked perisplenitis with adhesions, apparent atrophy of adrenal tissue, the enlarged spleen, and the marked lymphadenopathy, nothing of interest was found in the abdominal cavity." Examination of the material sent us was reported by Dr. F. W. Stewart as consistent with either pseudoleucemia or Brill's disease.

Comment. Marked palliation of a case of lymphatic pseudoleucemia following each of three radiation treatments of the entire body.

Case IV. *Myeloid leucemia.* Edward J., mechanic, aged thirty-five. Admitted to clinic March 1, 1932. History of pain and swelling in left upper abdominal quadrant of four months' duration; loss of 12 pounds. Examination essentially negative except enlarged spleen, extending 13 cm. below the costal margin in the midclavicular line and 11 cm. above the costal margin in the anterior axillary line. General condition good.

Blood count: Hb, 80 per cent; red blood cells, 3,904,000; white blood cells, 36,400; polymorphonuclears, 20 per cent; large lymphocytes, 10 per cent; small lymphocytes, 22 per cent; transitionals, 10 per cent; eosinophiles, 3 per cent; basophiles, 9 per cent; myelocytes, 26 per cent.

Treatment and Course. First total irradiation, March 8 to March 17, 1932, 166 hours of actual exposure, dose 112 r at 7.3 meters. At end of treatment there was no change in the size of the spleen, but the blood showed a decrease of white cells to 15,600, the other features of the count being about the same as before. One

month later spleen had become definitely larger, extending 18 cm. below the costal margin, and white cell count had risen to 31,600.

Second total irradiation, May 6 to May 11, 1932, ninety-five hours of actual exposure, dose 112 r at 5.4 meters. Two weeks later, although the white cell count had fallen to 18,000, the spleen showed a further increase in size, extending 20 cm. below the costal border. The patient's general condition remained good, however, and he continued at his work. Two months later the spleen was smaller and white cell count was 20,200. In October, 1932, but little change was seen except the presence of moderately enlarged inguinal nodes. The white cell count rose to 57,600.

Third total irradiation, October 8 to October 17, 1932, 152 hours of actual exposure, dose 150 r at 7.3 meters. Practically no change followed this treatment except that the white cell count fell to 21,000. In December, slight hemorrhagic tendency (two spots of purpura and slight epistaxis), and slight cervical and axillary adenopathy. Spleen remained large, 20 cm. below the costal margin.

In April, 1933, because of rise of white cell count to 80,000, the *fourth total irradiation* was given; April 24 to May 1, 1933, 148 hours of actual exposure, dose 112 r at 7.3 meters. At the end of this treatment the white cell count had fallen to 37,600. From then until the date of this report the patient did fairly well, continuing at his work, but the spleen remained large and the white cell count continued at higher levels than during the earlier course.

Later Note. Towards the end of August, 1933, because of evidence of progression of the disease (increased size of spleen, enlargement of liver, increased external adenopathy, and sharp general decline), local irradiation was given to the spleen, the Heublein unit having been closed for economic reasons. Definite symptomatic improvement resulted.

Summary. Definite improvement in the blood count followed each of four irradiations of the entire body in this case of myeloid leucemia, but there was no satisfactory change in the size of the spleen, such as usually results from local irradiation.

Case v. *Hodgkin's disease.* Martin B., student, aged sixteen. Admitted to clinic November 11, 1932. History of adenopathy in right axilla of two months' duration.

Examination. Well-developed, slender young man. General physical examination showed nothing of note except adenopathy. One firm movable node 2.5 cm. in diameter in right axilla. Below this was a recently healed biopsy wound. In left inguinal region was one enlarged node, 1.5 cm. in diameter. Biopsy (Dr. F. W. Stewart): "Probably Hodgkin's disease. Sternberg histology lacking. Very rare eosinophiles. Mostly reticulum hyperplasia and capsular fibrosis. Not certain of diagnosis." Blood count normal. Chest film negative. Basal metabolic rate +28 per cent; +22 per cent. Clinically the only tenable diagnosis seemed to be early Hodgkin's disease.

Treatment by local irradiation, using 200 kv. at 30 ma., distance 50 cm., with 0.5 mm. Cu filter, 500 r (measured in air), to right axilla, 300 r to left groin, 300 r to mediastinum anteriorly, and 300 r to mediastinum posteriorly, November 22 to December 1, 1932.

Result of local irradiation, almost complete regression of nodes by December 16. By February 2, 1933, patient had gained 8 pounds, and there was no adenopathy except two small shotty nodes in each groin and two small soft nodes in the right axilla.

Total irradiation (in accordance with the principle we had then established of giving a small dose of general irradiation to cases of Hodgkin's disease following the reduction of lymph node masses by local irradiation), February 6 to February 11, 1933, 83.5 hours of actual exposure, dose 75 r at 7.3 meters.

Course. March 1, 1933, patient declared a remarkable improvement in his health. Apparently there was further reduction in the adenopathy. Weight had increased 2 pounds more. Remained symptom-free up to time of this report (August 1, 1933).

Later Note. Last observation October 24, 1933. Further increase in weight of 5 pounds. Excellent general condition. No adenopathy, or other evidence of disease. Had returned to school. Blood counts remained normal except for one low white cell count (3,400) obtained on June 27, 1933. The white count on October 24, however, was 6,700 and the differential count had remained normal throughout. No other treatment has been given.

Comment. A case, clinically, of early Hodgkin's disease (although not substanti-

ated by the biopsy), in which a small dose, 75 r of total irradiation, employed after regression of enlarged nodes had been brought about by local roentgen treatments, was followed by marked general improvement. It is possible, of course, that this patient would have shown the same improvement without the Heublein treatment, but his case is cited as merely one of several in which a similar combination of treatments produced results that impressed us as superior to what we would expect solely from the amount of local irradiation that was used.

CASE VI. *Hodgkin's disease.* James K., postal clerk, aged twenty-three. Admitted to clinic July 30, 1932.

History. Right cervical adenopathy began three years previously. Three months before admission this swelling increased, and patient began to lose weight and strength and to have pains in right shoulder and chest. Nodes of right cervical region were removed three months before admission. They showed typical structure of Hodgkin's granuloma. Adenopathy soon recurred in the right neck. Loss of 22 pounds.

Examination. Well-developed and well-nourished young man in good general condition. No abnormal findings except adenopathy: numerous scattered nodes throughout lower right cervical region, largest being 1.5 cm. in diameter. In the upper cervical region was a group of nodes matted together, forming a mass 6×2 cm. There were small nodes palpable in the left submaxillary region. Within two weeks a node appeared in the left axilla and within six weeks large nodes were found in each groin. Chest film negative. Blood count normal.

Local irradiation, August 19 to September 22, 1932, 400 r (measured in air), using 200 kv., a 50 cm. distance with 30 ma., and 0.5 mm. Cu filter, to each of the following areas; each axilla, upper and lower mediastinal fields anteriorly and posteriorly, right and left cervical regions, and each groin.

Results of local irradiation: Marked regression, leaving only a soft fullness in the right upper cervical region. On January 19 and January 24, 1933, 400 r was given each time to this area.

First total irradiation, January 26 to January 30, 1933, 83.5 hours of actual exposure, dose 75 r at 7.3 meters. One month following Heublein treatment, March 1, 1933, had gained 9 pounds since admission to unit, and felt very much better. Adenopathy less.

Additional local irradiation: a single dose of 400 r to right upper chest posteriorly, because of pain in this region.

On March 29, 1933, patient's general condition was not as good, possibly as a result of continued hard work. The following month he had severe temporal throbbing headache for three weeks. Skull film negative. A diffuse swelling was found in left upper cervical region. Aside from this, and a few small nodes in right cervical region, there was no palpable adenopathy.

Second total irradiation, April 24 to April 29, 1933, ninety-nine hours of actual exposure, dose 75 r at 7.3 meters.

Course. For two days following discharge from Heublein unit was very weak and tired, appetite was poor and he was nauseated. After that, however, these symptoms disappeared, appetite became better than it had been before his admission to the unit. On May 16, 1933, no adenopathy except a few small soft nodes in left axilla. July 27, 1933, good condition. Only a few palpable nodes in neck and axillae; none in groins.

Later Note. October 5, 1933. Was given one local treatment of 400 r to right groin, because of the appearance in that region of one node 1 cm. in diameter. Otherwise patient was in excellent condition.

Last Observation. November 16, 1933, following continuance of hard work general condition had failed, patient had lost several pounds and was having generalized aching pains in back, shoulder and both lower extremities, although no adenopathy could be found.

Comment. Following each of two irradiations of the entire body this case of Hodgkin's disease showed general definite improvement.

CASE VII. *Multiple myeloma.* Florence B., housewife, aged thirty-three. Admitted to hospital November 9, 1931. Referred by Dr. A. C. Heublein. Symptoms of five months' duration. Roentgen film of skeleton showed typical lesions of myeloma in skull, humeri, ribs, vertebrae (partial collapse of 6th dorsal), pelvis and femora. Bence-Jones positive. Blood count,

secondary anemia (Hb, 70 per cent; R.B.C. 3,500,000). Patient barely able to get about, requiring several minutes to get out of bed, and having marked pain in legs, left and right chest and in mid-dorsal region. Abduction of upper extremities caused severe pain in dorsal region. Tenderness over dorsal spine.

Treatment and Course. On November 16, 1931, a single local roentgen treatment was given to the dorsal spine, from the 4th to the 11th dorsal vertebra. Dosage, 700 r; factors, 200 kv., 30 ma., at 50 cm., filter 0.5 mm. Cu.

First total irradiation, November 23 to December 10, 1931, 298 hours actual exposure, dose 375 r at 5.4 meters. Examination two months later, February 11, 1932, showed marked general improvement. Able to move about fairly freely, going up and down stairs with the aid of a cane, and being able to move arms about readily. She had practically no pain. Skeletal films showed slight increase in number and size of lesions, and the urine was still positive for Bence-Jones bodies. The anemia was more marked (Hb, 60 per cent; R.B.C., 3,008,000) and there was some leucopenia (W.B.C., 3,200).

Second total irradiation, July 7 to July 20, 1932, 238 hours actual exposure, dose 300 r at 5.4 meters.

At this time the general condition seemed very good. The blood count was essentially unchanged and Bence-Jones bodies were still present in the urine. Skeletal films showed no change. During this treatment period the patient had a severe attack of nausea and vomiting, lasting four days, but as similar attacks had occurred a few weeks before, it was felt that the irradiation was not responsible. Patient had no pain at this time.

She insisted on going home to Connecticut alone, although still unable to retain solid food. She failed to improve at home, and died August 27, 1932. Autopsy was not obtained.

SUMMARY

1. Heublein's method of total irradiation is original, differing from previously used methods in that roentgen rays are used continuously at low intensity, over periods of several days to three weeks, with target-skin distances of over 5 meters.

2. Experience with this method in 134 cases over a period of two years indicates that it is a valuable addition to the treatment of several radiosensitive tumor processes, such as the leucemias, lymphosarcoma, Hodgkin's disease and multiple myeloma, and that its results in the treatment of chronic lymphatic leucemia and pseudoleucemia seem, in our experience, superior to those obtainable by local irradiation. It appears to be of only slight value in the treatment of radioresistant tumors.

3. Certain difficulties in the use of the method are discussed, such as the development of leucopenia, thrombocytopenia, or anemia in some cases. Other temporary difficulties such as the failure to date to secure satisfactory regression of some local tumors, and the present empiricism in selection of dosage may be ascribed to the fact that there have been only two years of experience with this method. It is quite likely, therefore, that we have not as yet found the optimum dose, or the duration of treatment and spacing of treatment, which will yield the best results in any given group.

4. Various modifications of the method for future investigation are proposed: the treatment of restricted areas of the body by continuous prolonged short wave therapy, the use of small doses of general irradiation supplementary to local irradiation for the treatment of latent foci of radiosensitive processes, and the use of prolonged long-distance unfiltered or lightly filtered radiation for certain cutaneous diseases.

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AUDITORY EFFECTS OF ROENTGEN RAYS IN DOGS. II*

By EDWARD GIRDEN *and* ELMER CULLER

(*With cooperation of G. L. Clark*)

University of Illinois

URBANA, ILLINOIS

WE HAVE suggested the "conditioned response" technique as a means of direct approach to the study of the effects of roentgen rays upon hearing in dogs. Data on two preliminary animals indicated that irradiation with small doses generated at 85 kv. and 5 ma. resulted in a transient gain in acuity. Cumulated doses up to 4,400 and 11,000 r respectively failed to produce obvious auditory impairment.[1]

Several small doses applied at two day intervals (total 20 r) also resulted in a transient gain in acuity in a third dog (No. 3—June, 1932) which lasted for about four weeks. In this case, however, the change occurred on the third day after the application of the first dose. The short latent period suggested the possibility of an artefact in this dog. It was then decided to do all future testing with control dogs: i.e., dogs tested under identical conditions but *not* irradiated.

This study is a report of the results obtained from seven dogs which have been tested with controls. It might be mentioned that no control dog has at any time shown an increase in acuity. As in previous animals, *the area of irradiation* was the top of the head, and *included all of the auditory tracts*, peripheral as well as central. The smoothed data for typical cases will be indicated graphically since the raw data and their statistical treatment have appeared elsewhere.[2]

RESULTS

Dog No. 4. No. 4, male, and No. 5, female, were hounds of the same litter. The dogs were trained in September, 1932, and the first limina were obtained the following month. The tests were highly consistent, but the irradiation of No. 4 had to be delayed because of an unavoidable change in test conditions late in November. Incidentally, No. 5 had a litter of puppies November 11, but this event did not affect the limen.

Irradiation of No. 4 started December 13, and the dog was exposed on five successive days for a total of 39 r. The roentgen conditions are given in Table 1. The rise in acuity appeared on December 20, after a latent period of seven days. No. 4 was noticeably nervous and active from December 26 to January 5. No similar change occurred in No. 5, the control dog. The period of increased acuity lasted in No. 4 for about seventeen days. The possibility that the change in acuity was due to this increased nervousness was not substantiated in subsequent dogs in whom an increase in acuity occurred following irradiation but without the accompanying nervousness. All the limina of No. 4 during the period of increased acuity were significantly different from the pre-irradiation tests, indicating that the change was not a chance difference. Those of the control, No. 5, remained within the range of normal variability.

The data for the two dogs are represented graphically in Figure 1. The smoothed limina of No. 4 are represented by the black dots and those of No. 5, the control, by circles. Each dot and circle represents the value of the limen (hearing ability) on a given test (day). The means of the successive groups of limina are given only for the test animal, No. 4, and are the means of the *raw data*. These means are represented by the short horizontal bars. The lower section of the graph, "deviations from control," represents the difference between the successive pairs of the smoothed limina of the test and control dogs taking those of the control, No. 5, as zero.

Dog No. 5. This dog was irradiated the following spring (1933) on four successive days for a total of 60 r. The latent period was twelve

[1] Girden, E., and Culler, E. Auditory effects of roentgen rays in dogs. AM. J. ROENTGENOL. & RAD. THERAPY, 1933, *30*, 215–220; 539.

[2] Girden, E. Auditory Effects of Roentgen Rays in Dogs Doctoral Thesis, University of Illinois, May, 1934.

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TABLE I*

Animal	r/sec.	Length of Exposure	Total r Exposure	Special Conditions
No. 3				
June 2, 1932	2.5	2	5	
4		2	5	
6		2	5	
8		2	5	
		8 sec.	20 r	
No. 4				
Dec. 13, 1932	3.0	2	6	
14		2	6	
15		2	6	
16		2	6	
17		5	15	
		13 sec.	39 r	
No. 5				
Mar. 7, 1933	3.0	5	15	
8		5	15	
9		5	15	
10		5	15	
		20 sec.	60 r	
No. 3, 5				
Oct. 12, 1933	2.5	6	15	
13		6	15	
14		6	15	
26		20	50	
		38 sec.	95 r	
No. 7				
Oct. 26, 1933	2.5	20	50	
Nov. 10	2.8	12	35	Kv. peak: 100 kv., Nov. 10.
		32 sec.	85 r	
No. 8				
Oct. 12, 1933	2.2	5	11	Kv. peak: 200 kv. throughout.
13		5	11	2 mm. Al filter, Oct. 12, 13; 30
27	2.7	12	45	cm. target dist. throughout.
		22 sec.	67 r	
No. 9				
Oct. 12, 1933	2.2	45	100	Kv. peak: 200 kv. throughout.
13		90	200	2 mm. Al on all but last three
19		105	230	doses; 30 cm. target distance
20		120	265	throughout.
26		120	265	
26	3.7	120	445	
Nov. 2		300	1110	
10		240	890	
		1140 sec.	3505 r	
No. 11, 12				
Dec. 21, 1933	2.5	20	50	
22		20	50	
23		20	50	
		60 sec.	150 r	

* Unless otherwise specified under "Special Conditions" the following are to be understood: (1) 80–90 kv., (2) 5 ma., (3) 25 cm. target distance, (4) area of exposure: extends 8 cm. in front of lambdoidal ridge and entire width of skull, usually 13 cm.

days and the period of increased acuity lasted for about thirty-six days. The control animal (No. 3) did not show a similar change. The test animal showed signs of nervousness only during doses of 15 r each were applied to both dogs and were followed by a single dose of 50 r twelve days later. After a latent period of eleven days the acuity of both dogs increased for ten and

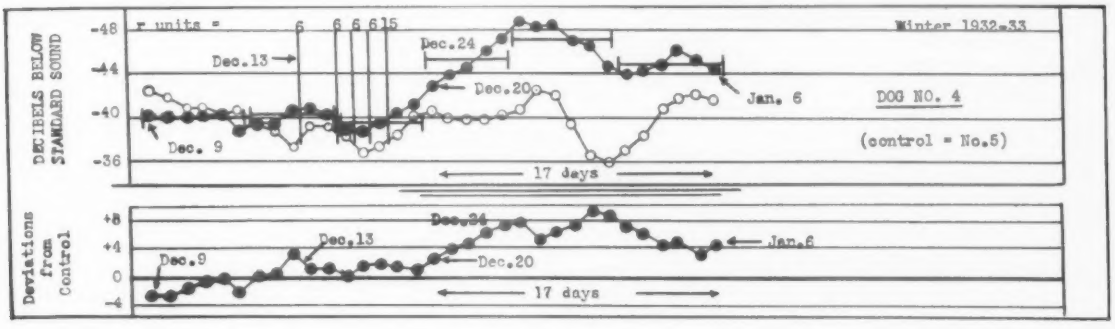

FIG. 1. Interpretation: The limina of the irradiated dog are represented by the dots, and each horizontal bar is the mean of the several limina within its range. The limina of the control dog are represented by the circles. The subsection, "Deviations from Control," indicates the variability of the test dog in terms of the control; the limina of the latter are taken in each case as zero.

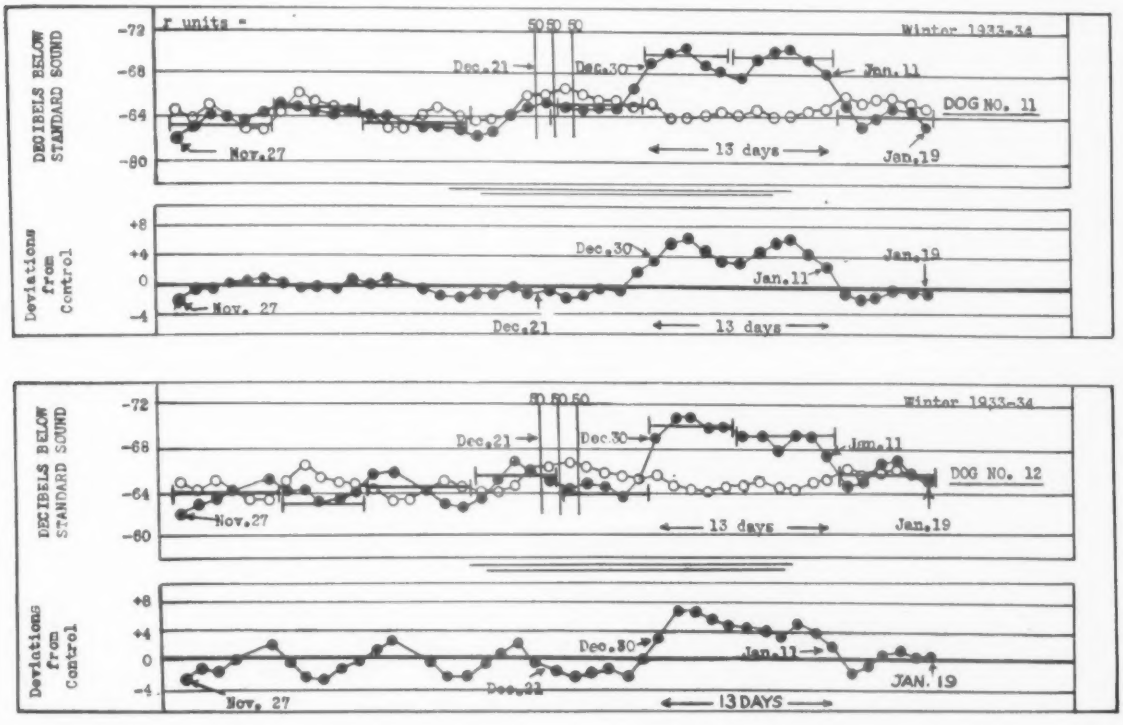

FIG. 2. Legend as in Figure 1. Dogs No. 11 and 12 were irradiated under identical conditions, and No. 6 served as control for both animals.

a three day interval during the period of increased acuity; at all other times the animal's behavior was entirely normal.

Dogs No. 3,5,7. During the Fall (1933) Nos. 3 and 5 were again irradiated. Three daily eleven days respectively. At the same time No. 7 was irradiated with a single dose of 50 r (Oct. 26). The effects appeared in this dog after a latent period of eleven days, and the increase in acuity persisted for twelve days. It is quite

likely that the effect lasted for a somewhat longer period of time in all three dogs, since they, as well as the control, all dropped at the same time due to a change in the test situation. The estimation of the gain in acuity for these dogs is therefore a bare minimum. No. 6 served as control for all three dogs, and showed no similar gain in acuity.

Dogs No. 11, 12. These two dogs were the last to be irradiated with roentgen rays generated at 80–100 kv., and No. 6 again served as control. The two test dogs were trained during the time the previous dogs were being tested (Fall, 1933). The first limina were taken in November. The test dogs were irradiated under the same conditions: doses of 50 r on three consecutive days beginning December 21. The rise appeared in both dogs December 30, nine days after the application of the first dose. The data of the two animals, as represented in Figure 2, are given in separate sections for the sake of clearness. Since No. 6 served as control dog for both Nos. 11 and 12, its smoothed limina (as represented by the circles) are identical in both sections. Again, the subsections, "deviations from control," represent the deviations of the limina of the respective test animals from those of the control, No. 6. Some signs of nervousness appeared in No. 11 on several occasions. No such behavior was noticeable in No. 12. The data of the control dog, No. 6, were consistent throughout: the limen was of the same magnitude as obtained in previous months. The period of increased acuity was the same for Nos. 11 and 12: thirteen days.

Dogs No. 8, 9. These dogs were used in a preliminary study of the effects of roentgen rays generated at 200 kv. and 5 ma. The area of exposure was the same as for the previous animals, but the target distance was increased to 30 cm. In most cases the beam was filtered through 2 mm. aluminum (Table 1).

No. 8 was an excellent test animal. This dog was irradiated October 12, 13, and 27 (1933), for a total of 67 r. Some slight changes appeared but for the most part were well within the limits of normal variability. To test the possible effects of large doses generated at the same voltage, more than 3,200 r were cumulated in No. 9 from October 12 to November 10 (1933). Unfortunately this animal was not a satisfactory test animal. The limina were not absolutely accurate and never within 5 decibels of the other dogs. The only developments that occurred were the appearance of skin burns and the loss of hair. The attempt to produce gross impairment of the auditory function failed, as it had with Nos. 1 and 2 at 80–90 kv. and 5 ma.

DISCUSSION

The mean gain in acuity for the eight dogs irradiated with roentgen rays generated at 80–100 kv., and 5 ma., under the conditions noted, was about 5.5 decibels, lasting from twelve to thirty-six days. Two of the dogs under daily irradiation showed a second, although shorter, period of increased acuity. This is supported by evidence obtained from two other dogs which were irradiated after the return to normal; the latter dogs too showed a second and shorter period of increased acuity. In every case the hearing returned to the pre-irradiation level, even with irradiation during the period of increased acuity. On the whole, the latent period was usually about eleven days; the two exceptions are Nos. 3 and 4. The results obtained with 200 kv. suggest that the shorter waves are not absorbed as much and hence have less (if any) effect. Large cumulated doses will produce no impairment of auditory function even if the doses are great enough to produce skin burns and loss of hair.

The clinical evidence is inconclusive. Desjardins[3] believes that the evidence on the whole is negative. Considering the number of pathological conditions that have been attacked, the variety of methods that have been employed in testing hearing as well as the number of different roentgenological conditions, it is not surprising that the results are not consistent. Our data would incline one to believe that irradiation of all of the auditory tracts by general irradiation of the head should have a beneficial effect, even if temporary in nature. Richardson has stated that just such an effect is produced under these conditions, though he claims that the relapse

[3] Desjardins, A. U. Action of roentgen rays and radium on the eye and ear. AM. J. ROENTGENOL. & RAD. THERAPY, 1931, *26*, 634; 787; 919.

(when it does occur) is not to the previous low level of acuity. These results were obtained from patients suffering with impaired hearing.[4] Since no data have been procured from dogs with impaired hearing we cannot say whether this phenomenon is limited to dogs with normal hearing

It is intended to pursue the investigation along several lines which will ultimately lead to an understanding of the mechanism involved in the increased acuity resulting from roentgen irradiation. The immediate steps to be carried out will be (1) to determine the locus of the effect, and (2) to work out the relationship between the size and spacing of the doses and the duration of the resulting period of increased acuity. The solution of (1) may be attained by differential irradiation of the central (brain) and peripheral (cochlea) mechanisms, or by direct study of the neural phenomena of the cochlea, eighth nerve and higher brain centers before and after roentgen irradiation. The former, at present, is the more accessible approach, but it is hoped to be able to develop practical techniques for the latter approach. Some work has already been done on (2), indicating that large doses result in shorter periods of increased acuity, but as yet the data are not conclusive.

[4] Richardson, J. J. X-Rays as an advance in the treatment of impaired hearing. *J. M. Ass. Georgia*, 1924, *13*, 161–165.

SUMMARY

It is noted that a temporary increase in acuity of about 5.5 decibels, lasting from twelve to thirty-six days, occurred in eight dogs following irradiation with small doses of roentgen rays generated at from 80–100 kv. and 5 ma., under the conditions noted. This betterment of *normal* hearing occurs usually after a latent period of eleven days. There is evidence in four of these dogs that subsequent irradiation will result in a second period of increased acuity, shorter in duration. Dogs which served as control animals and were *not* irradiated showed no similar betterment of acuity. The relationship between the dosage or its spacing and the size and duration of the gain in acuity has not been determined though there is some indication that the relatively larger doses result in shorter periods of increased acuity.

THE LEGAL ASPECT OF IDENTIFICATION AND INTERPRETATION OF ROENTGENOGRAMS

By I. S. TROSTLER, M.D., F.A.C.R., F.A.C.P.

CHICAGO, ILLINOIS

THE question as to whether roentgenograms may be introduced as (or in) evidence in court, with or without identification by the person who made them and whether they are of themselves the best evidence, and as such are to be interpreted by the (lay) jury, or whether they should or must be interpreted by experts, has come up for consideration and judicial opinion before so many of the higher courts in this country under so many varied aspects, phases and guises, and has been decided in so many different and dissimilar ways, that a review of some of the Appellate and Supreme Court decisions seems advisable and fitting.

It is of course primarily understood that our contention, from the standpoint of roentgenologists—and we believe that our ground is logical, tenable and absolutely correct—is that *all roentgenograms*, whether they be used in court or in clinical medicine, *should be properly and unqualifiedly identified* and that *the interpretation of all roentgenograms should be made by properly qualified medical roentgenologists and by no others*. Only a few of the State Supreme Courts agree to this.

Unfortunately, the law has in most instances looked upon roentgenograms as pictures—x-ray pictures—when as a matter of fact we know that they are not, nor cannot in any sense be considered as pictures at all, but are silhouette shadows of the structures portrayed in them. The courts holding and considering them to be pictures, further held in numerous instances that anyone can see, and seeing can entirely and completely understand what is shown in these pictures. It is a self-evident fact that this reasoning from false premises must in the natural order of events give false conclusions.

We could continue to argue along this line for the purpose of proving the correctness of our theory indefinitely, knowing as we do that we are correct, but *unless* and *until* we can convince the courts of this country of the correctness of our position, we may expect them to continue in their variance, one way today and tomorrow another.

Some of the highest state courts have decided that roentgenograms are inadmissible unless properly identified, and in the next breath they proceed to accept what we know to be very questionable identification. Other courts are stricter as to the identification, but so lax as to the interpretation that justice would surely be better served if the roentgenograms had not been admitted at all. Practically none of the accessible decisions are as strict regarding the identification before the admission in evidence and proper interpretation by competent experts as we know the importance of roentgenograms as evidence is and should be considered in the eyes of the law.

In our opinion, one of the soundest and best expressed opinions in favor of our line of argument regarding identification is a Texas case,[1] where the attorney for the plaintiff took the witness stand for his client and testified that the day before he had caused a roentgenogram of the plaintiff's injured leg and ankle to be made by a physician, at the same time offering the roentgenogram so made in evidence. The attorney testified that he had seen the roentgenogram made; that the physician who made it was competent to make it; that of his own knowledge the roentgenogram was properly and correctly made and that it showed the condition of the bones of the leg and ankle as they then existed.

Of course the opposing attorneys ob-

jected to such testimony and evidence as incompetent, but the trial court admitted it over their objections and a verdict and judgment for the plaintiff resulted.

The case was appealed by the defendants and the Court of Civil Appeals of Texas in deciding the case said, that it was "**Unable to see how the attorney for the plaintiff would or could be competent to testify as to the professional skill of the physician* or upon what logical basis he was competent to judge of the skill of the physician who made the roentgenogram" and that "The testimony and evidence was incompetent and inadmissible" and remanded the case for retrial.

While this Texas case decision does not in so many words say that the roentgenograms must be identified in a certain manner, it is considered and quoted in so many other decisions that it appears to go farther and carry more weight than any we have been able to discover.

In some states, courts have established precedents that objects—particularly pictures, including roentgenograms—must be properly indentified before they become competent evidence, and in the case of objects concerning which expert testimony must be given for their proper interpretation, the person or persons identifying the objects must likewise be competent or the objects themselves remain inadmissible.

On April 16, 1924, I presented a paper before the Chicago Medical Society (published in *Illinois Medical Journal* for August 1924) entitled "An Important Supreme Court Decision." In that paper I discussed a case[2] wherein a dentist had identified a roentgenogram made by him and where the Supreme Court of Illinois had said:

> The most serious error committed by the trial court in the admission of incompetent evidence, was the admission of a so-called x-ray film which was purported to show the condition of the plaintiff's skull. A dentist identified a film produced in court as one prepared by him, and testified that it was an x-ray picture (sic) of the plaintiff's skull.

* Italics mine.

The court said:

> Although a skiagraph produced by x-rays cannot be verified as a true representation of the object in the same way as a picture made by a camera, the rule in regard to the use of ordinary photographs in the trial of a cause applies to skiagraphs of the internal structure and condition of the human body taken by the aid of the x-rays, and such a skiagraph, when verified by proof that it is a true representation is admissible evidence. Like other photographs they cannot be received as evidence until proper proof of their correctness and accuracy is produced.

The case was remanded because the roentgenogram was made and introduced without the maker having properly qualified, and the introduction of the roentgenogram was not predicated upon the fact that it was a true representation of the condition as measured by the requirements of the court and the law.

In a much quoted Oklahoma case[3] the Supreme Court of Oklahoma said, referring to roentgenograms:

> The plates were not admissible in evidence nor could they be, until they were properly identified and shown to be made by a trustworthy instrument properly used by a person skilled in making, reading and interpreting such plates, and further showed to be correct representations of the bony structure of the plaintiff's head. The admission of the roentgenographic plates in evidence rests fundamentally on the theory that they are the pictorial communication of a qualified witness who uses this method of conveying to the jury a reproduction of the object of which he is testifying; this being true, the roentgenographic plates must be a part of the testimony of some qualified witness, and the witness should qualify himself by showing that the process is known to himself to give correct representation, and that it is a true representation of such objects.

In a leading Iowa case,[4] the Iowa Supreme Court held almost identically the same opinion as in the foregoing Oklahoma case, and in another case,[5] the same court decided exactly as it did in the Matheson case but adding "It was error to permit a physician to testify to what appeared in a roentgenogram."

In an action for damages resulting from injuries to the plaintiff's leg from a fall upon a defective sidewalk, it was held by the Court of Civil Appeals of Texas[6] that it was not in error to refuse to permit the defendant's expert to testify as to what appeared in the roentgenogram of the injured leg, in view of that court's previous holding that the roentgenograms themselves are the best evidence of what appears in them.

The court said:

It is proper for an expert to explain any x-ray photograph in such particulars that are not understood by a layman (sic). What the jury could see and understand about the matter is not the subject of expert testimony and this we understand to be the effect of our prior decisions. A radiograph may be used for purposes of demonstration by an expert, as though he had the object itself before the jury for explanation.

That the bones can be distinguished from the flesh in an x-ray photograph and that the bones would make a heavier shadow than the muscle is proper expert testimony. Such scientific facts would not be known to the average layman.

Again in another case,[7] the State Supreme Court of Iowa, in affirming a judgment for the plaintiff, stated almost identically the same words as in the last mentioned Texas case.

The Court of Errors and Appeals of New Jersey evidently believes that roentgenograms must be properly identified; but that having been done, anyone may interpret them, though this is not expressed in so many words. In affirming a judgment for the plaintiff for damages for personal injury[8] that court said:

The defendant contended that there was error in admitting in evidence certain roentgenographic plates and films, and in allowing them to be taken into the jury room. But the court held that if the correctness of the roentgenographic photographs (sic) is established by the testimony of the person making them, or if they are identified by the surgeon under whose general direction and for whose use they were made, and by whom they were used in his diagnosis of the bodily injury, they are admissible in evidence and may properly be taken into the jury room.

The Court of Appeals of Kentucky, in affirming a judgment in favor of the plaintiff[9] said:

The defendant city prosecuted its appeal chiefly, if not solely, on the ground that certain roentgen ray pictures (sic) . . . were allowed to be introduced without sufficient qualification of the witnesses by whom they were introduced and without sufficient verification of the photographs. . . . The court holds them to have been properly verified . . . The rule with respect to the introduction of roentgenographic photographs is the same as that governing the introduction of ordinary pictures.

A decidedly slender and weak identification was accepted and approved by the Supreme Court of Oregon,[10] which said:

In this action to recover damages . . . the defendant contended that the trial court erred in admitting some roentgenograms of the plaintiff's broken bones, on the ground that the roentgenograms were not properly identified. However the physician under examination at the time that the roentgenograms were introduced in evidence, declared that they were pictures of the injured part. That was sufficient to overrule the objection, which, indeed, went only to the weight of the testimony.

In a comparatively recent Illinois case, the question of the admissibility of roentgenograms made on an old machine entered into the arguments. In this case[11] a roentgenologist testified that he had personally exposed and developed certain roentgenograms of the cervical spine of the plaintiff, but that the apparatus he had used was old, antiquated and not in very good working order. The roentgenograms were correct representations of the plaintiff's cervical spine according to this physician roentgenologist's testimony.

Defendant's counsel contended that under the circumstances stated, the roentgenograms were not competent evidence. The jury brought in a verdict for the plaintiff, judgment was so entered and the defendant appealed.

The Supreme Court of Illinois, in rendering a decision sustaining the judgment of the lower court, said:

In the absence of convincing proof to establish accuracy, it would certainly be improper to admit roentgenograms shown to have been taken on a machine not in good working order; but no absolute rule has been laid down making roentgenograms taken on such a machine inadmissible, regardless of other testimony and conditions. In the present case the evidence was properly admitted.

It might very properly have been added that in the absence of other roentgenograms or other evidence which would show the condition of the cervical spine in this case, these roentgenograms were the best evidence obtainable in the circumstances and as such "best obtainable evidence" were and should be admitted.

The Supreme Court of Missouri, in affirming a verdict of the lower court in a personal injury case,[15] said:

In this action . . . a physician testified that he was a roentgen-ray specialist, had examined the right arm of the plaintiff with the x-rays, and had the x-rays that he made at that time. The plaintiff offered the roentgenograms in evidence in connection with the testimony of another physician. Objection being made that they had not been sufficiently identified. The plaintiff did not press his offer but later recalled the roentgenologist. The latter testified that one of the roentgenograms was a roentgenogram of the plaintiff s right forearm: that it bore the title "Mr. Mattice, File 17927, Nov. 1922," which was the date the roentgenogram was made, adding "I didn't examine the hand myself, any more than just the x-ray; no physical examination at all." On cross examination he testified that he took the pictures himself, but did not do the development of them himself; that "you get them out of the dark room, and you send them back, and watch them after they are developed and check these things all through the process. Although I wasn't in the dark room, I know that this is the same plate that I took of the plaintiff's arm because you go in there as soon as they are in the hypo; you see these things before the process of silver elimination is complete. . . . I am positive that this is the plate that I took, although it passed out of my hands, and stayed a while in the dark room." Again he testified: "These pictures were developed immediately after the exposures were made, before the man leaves the office. They are checked up before he gets out. We do not have more than one patient's plates in there at a time, that is, being developed at the same time. We take a patient and finish him up, especially on work of this sort. . . . I have an idea that we did have some other broken arms that same morning, but that is a roentgenogram of this man's arm." Such being the testimony, and the defendant standing on its objection to the introduction of the roentgenograms in evidence, plaintiff's counsel withdrew his offer of the roentgenograms.

The roentgenologist had also been examined at length concerning what was shown by the roentgenograms, and at the close of the evidence defendant's counsel asked the trial court to exclude the roentgenologist's testimony and to instruct the jury to disregard it, on the ground that his testimony was based on plates that he held in his hand and from which he testified, which had not been sufficiently established to justify him in testifying from them. The trial court refused that request; and the Supreme Court holds that was not error, under all the circumstances. The trial court did not have an opportunity to rule directly on the admissibility of the roentgenograms, because the plaintiff withdrew his offer of them, but the Supreme Court concludes that there was sufficient identification of them to make them admissible in evidence, and to make them the proper basis, as the trial court held, of the testimony of the roentgenologist.

In an action[16] to recover damages resulting from being injured by a truck, the defendant contended that the trial judge had improperly admitted in evidence certain roentgenograms showing the character of the injury. This objection was based on the fact that the physician who produced the plates at the trial had not himself taken them. The plates, however, were taken by another physician, under the supervision of the one who testified, and at a hospital at which the witness was engaged in professional work, and to which the injured child was taken.

The Court of Errors and Appeals of New

Jersey in affirming the verdict of the trial court in favor of the plaintiff, said:

The rule is laid down in the case of Robinson vs Payne,[8] that if the correctness of the roentgen-ray photographs is established by the testimony of the person making them, or if they are identified by the surgeon under whose general direction and for whose use they were made, and by whom they were used for his diagnosis of the bodily injury, they are admissible in evidence.

This court is therefore of the opinion that the roentgen-ray photographs were properly admitted in evidence, as they were identified by the physician at the hospital to which the child was taken, and were made under his supervision and direction, and for his diagnosis of the injury.

While the circumstances surrounding the following case[17] resemble the weather in California in being "Most unusual," we are sure that it will be of interest and value, because of the clear and terse expression of the rule in regard to the admissibility of roentgenograms.

In a prosecution for murder, a coroner's physician testified that he had examined the body of the murdered man after death, that a roentgenogram showed that the bullet had passed from the left side to the right side of the body, shattering the ninth rib and the spinal cord, and that he only viewed the body and took the records from the hospital, but made no autopsy.

He admitted that there was some doubt as to the cause of death, but stated that in his opinion death was due to heart disease, superinduced by nephritis, an infection of the kidneys, and an infection of the bladder, due primarily to and caused by the cutting of the spinal cord by the gun-shot wound. No objection was made to the admission of this testimony when it was offered. On cross examination, however, when the witness admitted that he had made no post-mortem examination of the body and that he got his information as to the cause of death from the records of the hospital, counsel for the defendant moved to strike out his testimony with reference to the cause of death. The court did not rule directly on the motion but said that the witness might testify as to what he saw and remembered about the roentgenogram, which was "simply a physical thing." The roentgenogram was not introduced in evidence nor in any way identified, nor did the witness qualify as an expert in the reading of roentgenograms.

The defendant was convicted and appealed to the Supreme Court of Illinois, which in its reversal of the judgment of the trial court, said:

The testimony of the coroner's physician, when given from the records of a hospital, without proof of their authenticity, was clearly inadmissible, as hearsay, and should have been stricken out. His testimony concerning matters disclosed by the roentgenogram was likewise inadmissible because no ground had been laid for such testimony.

The rule in regard to the use of roentgenograms, when it is sought to put them in evidence requires that the witness testify either that the roentgenogram shows accurately what the witness saw by examining the body through a fluoroscope, or that the witness is skilled in the use of the roentgen machine, that he took the roentgenogram concerning which he testifies, and that he is able to say that it is accurate.

It was clearly error to permit the witness to testify as to his conclusions as to what the roentgenogram showed, without proof of his qualifications and of the accuracy of the roentgenogram, and without accounting for the failure to produce it. There was no proof of the cause of death, other than the testimony of this witness. As such proof rested on incompetent testimony, its admission was an error requiring the reversal of the judgment.

In a suit[18] to recover damages for personal injuries, in which a verdict was given to the plaintiff, the defendant appealed on the ground that a physician who had treated the plaintiff had testified concerning a roentgenogram showing the injuries incurred by the plaintiff. The roentgenogram had been made by another physician, who did not testify in the case but the roentgenogram in question had been made on the order of the physician who was testifying.

The objection was that "No foundation had been laid for the introduction of the roentgenogram." The Supreme Court of Kansas ruled that "There was no reversible error committed in the admission of evidence" and confirmed the judgment of the lower court.

The Supreme Court of Illinois, in affirming a judgment in favor of the plaintiff in a personal injury suit,[19] said:

> During the examination of a physician, the plaintiff offered in evidence two roentgenograms. When the first was offered there was an objection that it was not properly indentified. The physician had testified that he was present when the exposure was made, and that he waited while the plate was taken into the dark room and developed; that he examined the plate and was positive that it was the roentgenogram taken of the hand of the plaintiff. It was sufficiently identified and the objection was properly overruled. But the objection made when the second roentgenogram was offered in evidence should have been sustained, the objection being that a proper foundation had not been laid for its admission, for the reason that the witness did not develop the plate nor did he see it developed, nor could he testify that it was an accurate portrayal of condition of the bones of the hand. The error in admitting this roentgenogram, however, was not prejudicial, for the reason that the physician testified that the condition shown in this roentgenogram was the same as that shown by the other. Furthermore the physician testified fully concerning the condition of the injured hand, and there was no contention on the trial that the condition described by him did not exist nor that the roentgenograms showed a condition different from that described by him.

A hair-splitting decision, is it not?

The Kentucky Supreme Court[20] stated:

> Where there is no testimony to identify roentgenograms introduced in evidence as roentgenograms of the injured parts of the body that they are supposed to represent, they are inadmissible without the testimony of the persons who took them. In the present case however, two roentgenograms were made of the same injured limb. The second roentgenogram, with the exception of some minor particulars, showed the same character of injury as the first roentgenogram, to the admission of which exception was taken. The physician who took the second roentgenogram testified that his experience enabled him to swear that the roentgenogram to which exception was taken was a roentgenogram of the very same limb that was shown in the second roentgenogram, identified by him. According to this witness, the same bones of the human body are differently shaped and constructed in each individual, just as are the faces of such individuals. Moreover, the plaintiff and some of her witnesses testified that the roentgenogram in question was taken of her limb, that the doctor who took it immediately delivered it to her, and that she preserved it. But if such reasons were not sufficient to remove the objections to the introduction of the roentgenogram to the admission of which exception was taken, its admission could not be considered prejudicial material, since its only purpose was to prove the extent of the plaintiff's injuries, and the testimony was uncontradicted on that issue and no sort of earnest attack was made on the amount of the judgment.

The question of the conditions under which roentgenograms may be introduced as evidence, is quite comprehensively discussed by Chief Justice Miller of Kentucky in another case[21] which was decided by the same court, and by the New York Court of Appeals in still another case.[22]

The use of unverified notes on the interpretation of roentgenograms was disapproved by the Appellate Division of the New York State Supreme Court,[23] in an appeal from an award rendered by the Industrial Board of Compensation, under the Workmen's Compensation Law. The decision read:

> Award reversed, and matter remitted with costs against the State Industrial Board to abide the event on the ground that the finding that the claimant on the date of the accident was suffering from a tuberculous condition, which became activated by an accidental injury, was based on certain notes interpreting roentgen ray pictures made by a physician who was not called to verify the pictures or the notes and was therefore grounded on other than legal evidence.

In another Illinois case[24] certain roentgenograms were used as the basis of the testimony of some of the witnesses with respect to the extent of the disability of the claimant for compensation, but a proper foundation was not laid for the use of the roentgenograms and the Supreme Court of Illinois said that it was error to permit the introduction of this testimony.

The Supreme Court of Oklahoma[25] in affirming a verdict of the lower court, in favor of the injured person, and discussing the admissibility of certain testimony said, in part,

> The admission of x-ray plates in evidence rests fundamentally upon the theory that they are the pictorial communication of a qualified witness who uses this method of conveying to the jury a reproduction of the object of which he is testifying; this being true, the x-ray plates must be made a part of some qualified witness' testimony and the witness should qualify himself by showing that the process is known to himself to give correct representations, and that it is a true representation of such object."

The St. Louis (Mo.) Court of Appeals[26] decided that roentgenograms introduced by the plaintiff's physician, who did not make them but who was present when they were made, were sufficiently identified.

In a Delaware case[27] the admissibility of testimony relative to roentgenograms—which while in existence and obtainable—was objected to and by agreement was allowed, dependent upon the proper introduction and admission of the roentgenograms later in the trial.

In the course of the trial of another Iowa case,[28] the plaintiff's physicians undertook to interpret a properly identified and introduced roentgenogram to the jury. Defendant's counsel objected, asserting that this was not proper testimony and that the roentgenogram itself was the best evidence of what it showed. The objection was overruled. The jury rendered a verdict in favor of the plaintiff and defendant appealed to the Supreme Court of Iowa and was sustained.

This same Iowa Supreme Court[29] held that a photograph was itself the best evidence of what appeared in it, and in another case[5] it applied the same rule to a roentgenogram offered in evidence to show a curvature of the spine, the question being only whether it showed as a straight line or a curved one (sic). In the latter case this court held that the roentgenograms served a purpose similar to a photograph, but we know, of course, and fully realize that that assumption is not true in any sense, because without the explanation of how the spinal curvature shown in the roentgenogram varies from the normal, it could mean nothing.

But in another Iowa case[7] the same Supreme Court apparently again awakened to the importance of roentgenograms as evidence and opened the door to admit expert evidence in their interpretation by saying:

> It is proper for an expert to explain an x-ray photograph in such particulars as are not understood by a layman. . . . What the jury could see and understand about the matter is not the subject of expert testimony, and this we understand to be the effect of our prior decisions. A radiograph may be used for purposes of demonstration by an expert as though he had the object itself before the jury for explanation.

From a Vermont case[14] we read that:

> *The Supreme Court might properly take judicial notice of what is well known to the medical profession, namely, that a roentgenogram does not necessarily or ordinarily interpret itself to the observation of a non-expert. A roentgenogram carries various lights and shadows, the significance of which is known to the expert and not known to the non-expert.* (The italics are mine and are here introduced to show that some courts were really beginning to see the importance of this subject.)

In a case[30] appealed to the Supreme Court of North Dakota, the decision reads:

> There were introduced in evidence in this personal injury case, in behalf of the plaintiff, two roentgenograms, one of a portion of the plaintiff's spine, and the other of one of his feet. The physician who took these roentgenograms was placed on the stand as a witness for the

plaintiff. After identifying the plates, and stating that they were in the same condition as when they were taken, he was permitted to interpret and explain them. He was also permitted to give his opinion as an expert as to the nature and extent of the injuries as disclosed by such plates. It was contended by the defendant that the rulings incident to the reception of all this evidence were erroneous. But the court deems it proper to say that it was not apparent that the trial court erred in admitting the roentgenograms in evidence. Nor does this court believe that the court erred in permitting the physician to explain the roentgenograms. One of them showed a dislocation or subluxation of a vertebra. The other showed a fracture of a small bone in one of the plaintiff's feet. While it is true the roentgenograms themselves were the best evidence of what they showed, it is equally true that *a roentgenogram may wholly fail to convey to an ordinary layman the facts as they are actually there shown; and the court believes that it is proper for an expert to explain a roentgenogram in particulars that are not understood by a layman.* Nor does this court believe that the trial court erred in permitting the physician to give his opinion as an expert as to the nature and extent of the injury, and its probable duration and effect. The authorities generally hold these matters to be a proper subject of expert testimony. In short, the court holds that where roentgenograms have been proved by a physician, it is competent for him to explain them to the jury in particulars that are not understood by laymen; also, that it is proper to receive the opinions of medical experts as to the nature and extent of personal injuries.

The foregoing is one of the most favorable of all the decisions reviewed, toward our contention. It is too bad that more state courts of last resort do not promulgate such sane and sensible decisions. This one should be used as a reference and made to be a leading case. We should call the attention of our legal friends to it, at every opportunity.

A case[31] wherein the trial court admitted a roentgenogram in evidence and a deposition giving the diagnosis and interpretation of the same was also admitted, the defendant contended that this was improper and should have been excluded. The Supreme Court of Albama, in affirming the judgment of the lower court, said:

This testimony tended to show that the roentgenogram was taken by a qualified expert; that it was read by a witness qualified by experience and training to do so, and that it disclosed a fracture of the second lumbar vertebra. Objection was made to the roentgenogram on two grounds—(1) that it was not sufficiently identified as the roentgenogram so taken and testified to by the witness, and (2) that the reading of a roentgenogram is the work of an expert—a jury cannot read it (sic), and it should be admitted in evidence only along with the testimony of a competent reader of such roentgenograms, pointing out to the jury, the features showing the injury in question.

But the taker of the roentgenogram testified to an identifying number, placed by him on it, with the date. The roentgenogram was made a part of the record. An examination disclosed the number "490", and "6-4-23," standing for June 4, 1923. These marks, in connection with the testimony, were sufficient on the question of identification.

Members of this court, with the aid of such background of light, and probably with about the same skill available to the jury, examined the roentgenogram, but could not without aid, discover with any assurance the particular appearances indicating the fracture testified to by the expert witnesses, and were persuaded that roentgenograms may be much more valuable evidence when explained to the jury by a person skilled in reading them, giving the angle at which they were produced, and the special appearance of the part involved, indicating injury or not.

This court thinks, however, that this roentgenogram was admissible in evidence in connection with the expert testimony given by deposition. At least it showed that there was a roentgenogram, giving, with all the accuracy of nature's laws, certain outlines. This fact was a circumstance from which an inference might be drawn favorable to the opinion given by those entirely familiar with the details of the human anatomy, and skilled in detecting injuries thereto, by making and reading such roentgenograms.

Any inability of the jury to discover unaided the evidences testified to by the experts would probably weaken the probative effect of the

roentgenogram with the jury, and work no injury to the defendant.

In affirming a verdict for the defendant physician, in a malpractice case,[32] the Supreme Court of Oregon held:

> The testimony of a physician that certain roentgenograms . . . supported his diagnosis, when such roentgenograms were not produced at the trial. The Supreme Court held that there was no error in admitting this testimony since a proper showing was made that the roentgenograms were lost. The court held that the roentgenograms themselves were admissible as evidence only when supported by the testimony of the party making them or an expert who knew them to show accurately the true condition of the objects represented thereby. They are valuable only when supported by the testimony of a witness. . . . It is competent for one to have his expert in such matters explain roentgenograms under proper supervision, so that the court and jury may view the picture as the witness does.

The interpretation of lost roentgenograms by a roentgenologist, who appeared as an expert witness, was admitted in a case appealed to the Supreme Court of Missouri;[33] and in another case[42] decided by the same Supreme Court, the testimony of a chiropractor radiologist (?) regarding roentgenograms, was accepted, when the roentgenograms themselves were not in evidence.

In an unusually comprehensive decision, the circumstantial authentication of a roentgenogram is made to be admissible evidence, when the roentgenogram is lost, by the Supreme Court of Appeals of West Virginia. Here this learned Court says:[34]

> In this personal injury case, the roentgenologist who made a roentgenogram of the plaintiff s hip, and another physician examined the plate after it was developed, and determined from the information received from the plate and from an examination made of the injured leg that there was a fracture of the hip bone. After that the plate was mislaid and it could not be found at the time of the trial, although thorough search was made for it. Still these physicians were permitted to testify that it disclosed a fractured hip bone. The defendants contended that this was error, first because it was not shown that the roentgenogram was so taken as to represent correctly the object sought to be photographed; and, secondly, the roentgenogram itself being secondary evidence and it being lost, no testimony could be offered based on a view of it. But the court holds that there was no error in either respect.
>
> It is quite true that before a roentgenogram can be used as evidence it must appear that it was so taken as to represent correctly the object reproduced. Of course, it is not meant by this that some one must testify that it is in fact a correct representation. Manifestly, this would be impossible, for the reason that the object thus photographed is not visible except with the aid of the roentgen-ray machine. What is meant is that it must appear that the machine was so placed as to reproduce correctly the object brought within the radius of its activity. Nor need this necessarily appear to be a direct statement of the operator to this effect, although it may be, and ordinarily is, shown in that way. It may, however, as well be shown from a detail of the circumstances. In this case the physician who took the roentgenogram was shown to be an expert with large experience in this work. The picture was taken with a view of determining the nature and extent of the plaintiff s injury with a view to treating it, and treatment was administered for the relief of the injury which the roentgenogram disclosed, with favorable results. This might not prove absolutely that the plaintiff was injured as disclosed by the roentgenogram, but the fact that the injury sustained by her responded to the treatment ordinarily administered for the relief of an injury such as was shown by the roentgenogram was, to say the least, very strong evidence that her injury was of that character, and sufficiently authenticated the roentgenogram to justify its admission in evidence.
>
> Was it proper for the two physicians who examined this roentgenogram to testify as to what it indicated? It seems very generally held that, when the original of a document is lost, a copy made from a copy may be introduced in evidence on a showing of the correctness of the first copy and the loss thereof. It is the best evidence obtainable. In this case, if we treat the roentgenogram as but a copy of the then existing condition, we have the original, which was the condition of the bone at the time, lost by a change in the conditions due to a lapse of time and the treatment administered, the only

reproduction of that condition is lost, and the best evidence obtainable as to what condition actually existed at that time is the impression made on the minds of the two physicians from their examination of the plaintiff s hip and the roentgenogram thereof. It was not error to admit this evidence.

Furthermore, this court holds that where, after the trial of an action to recover damages for injuries sustained from a broken bone has been entered on, the defendant requests that a roentgenogram be made of the injured member, and the plaintiff is willing that it should be done it is not error for the court to refuse such request where no reason is given for not having made the same before the trial, and it does not appear how long the trial will be delayed by granting such request.

On the other hand, expert witnesses may not testify as to what a roentgenogram shows when the same is obtainable and not in evidence. The Kansas Supreme Court in its decision of a case[35] appealed to it said:

A physician may not testify concerning what was shown by a roentgenogram that is not produced in court.

The contention that the roentgenogram is a part of the hospital records and cannot be taken from the hospital presents no adequate excuse for a failure to produce it. There is no rule of law that places hospital records in a privileged class. They can be produced in court the same as the records of a corporation, or any other records, corporate or private, that may be necessary to further justice between litigants.

The roentgenogram should be produced in evidence so that the other party may use it for the purpose of cross-examination and submit it to its own medical experts for examination, to enable them to testify as to their interpretation if they disagree with the expert who interpreted it on behalf of the party relying on it.

Along the direct line of the reasoning in the quotation from the decision of the Kansas Supreme Court just stated, attention should be here called to the fact that not only are hospital records privileged communications (from another legal aspect, however), but that they as well as roentgenograms have been held to be "privileged" by the Supreme Courts of several states,[36,43,44,45] just as are personal communications from patients to their physicians.

Another angle of the situation is presented in a decision from the Appellate Division of the Supreme Court of New York in affirming a judgment[37] in favor of the plaintiff on account of an inujry, when it said, "There was error in allowing a roentgenologist to testify from notes concerning a roentgenogram examined by him; but which he had not made nor caused to be made." The roentgenogram had not been introduced nor had the persons who made same been called as witnesses. This witness testified that the roentgenogram which he had examined had on it the name of the plaintiff and a number. He testified that he had examined it and made notes and a record of his findings and he testified therefrom. Defendant had entered objections to the admission of this testimony but the trial judge admitted it, and defendant's motion to have the testimony expunged was denied. The Appellate Court, however, said, "This was all wrong."

In a leading New York case,[71] one of the errors alleged by the defendant in the appeal related to the failure of the plaintiff to produce roentgenograms concerning which expert witnesses for the plaintiff testified. The Appellate Judge who wrote the decision, confirming the judgment of the trial court, said:

A careful reading of all the medical evidence as to injuries will show that the plaintiff was substantially injured. One of the plaintiff s witnesses, Dr. Gardner, swears: "I was present when the (X-ray) pictures were taken, saw them taken, saw them developed, and looked at the plates immediately after they were developed." Thus their identity and correctness were established. Then the Doctor proceeds to tell what the pictures show. Another physician, Dr. Tinker, also tells what he thinks the plates show. There was no demand made by the defendant for the production of the plates; no hint or suggestion that they were desired. They were in fact, in court, so the plaintiff states in his brief. I can see no error at all in this X-ray

incident. *The evidence shows that nobody but an X-ray expert could tell anything from the plates, and if they had been introduced they would have done the court, jury and the defendant's ordinary physicians no good. I do not think that the doctrine that an ordinary photograph is the best evidence of what it contains should be applied to X-ray pictures. They constitute an exception to the rule concerning ordinary documents and photographs, for the X-ray pictures are not, in fact, the best evidence to laymen of what they contain. Generally, they are no evidence at all, signifying nothing whatever except to the expert. The opinion of the expert is the best evidence.* (Author's italics).

If there had been a demand for these pictures in court, or a request that they be submitted to the inspection of the opposing experts, and these rights had been denied the defendant, such an error would be serious. But whatever harm befell the defendant, if there was any harm at all, came to it by reason of its own failure to demand what it was entitled to, and what it would have unquestionably have received by the mere asking.

From the roentgenologist's standpoint, all we need to do is to call attention to the italicized portion of the foregoing excerpt from this decision and to stress that as our argument.

In addition to stating what the evidence shows, the learned judge who wrote the decision evidently agrees entirely with our contention and believes what we claim and know to be the truth.

The competency of a witness to interpret roentgenograms was decided as we believe it should be in a case appealed to the Supreme Court of North Carolina[38] in which the court said:

Whether a witness is competent to testify as an expert is a question addressed primarily to that sound discretion of the court, whose decision is ordinarily conclusive.

The testimony of a witness explanatory of a roentgenogram was properly excluded in a finding by the court that he was not sufficiently qualified to express an expert opinion on questions of anatomy in connection with the proposed explanation.

This is certainly one of the first clear cut demonstrations that the courts of this country are becoming alive to facts which we as roentgenologists have known and contended were right, just and proper, for many years.

Allowing roentgenologists to show roentgenograms of normal persons for comparison has brought out some decisions. The Supreme Judicial Court of Massachusetts, in overruling the defendant's exceptions in an action[39] to recover damages for personal injuries, for which the plaintiff obtained a verdict, said:

A physician who was a specialist in the use of the roentgen rays was called as a witness by the plaintiff, and testified concerning certain roentgenograms purported to be of the plaintiff's pelvic region, where the principal injury claimed by her existed. He said that the plates showed an abnormal condition of the pelvis, particularly with reference to the relation of the bones, some of which were alleged to have been fractured. He also produced another roentgenogram, which he testified showed the pelvic bones of a normal person, this plate being admitted in evidence subject to the defendant's exception.

It is settled that a roentgenogram showing the physical condition of a plaintiff in an action for personal injuries is admissible in evidence, and this court sees no valid reason why a plate of another person, properly verified by proof, which showed the pelvis in its normal condition, was not competent to assist the jury in understanding the nature and extent of the plaintiff s injury, in the same way that a skeleton is often exhibited in similar cases. Whether the plate was sufficiently verified was a preliminary question of fact to be decided by the trial judge, and was not subject to exception. As the plate in question would have been of assistance to the jury in understanding the case, this court is of the opinion that it was admissible in the discretion of the trial court.

A physician called by the defendant testified that he had examined the plaintiff at the hospital. He described the injuries which he found and stated that there had been fractures of two bones of the pelvis, which were well set, and that the pelvis was "tipped a little bit." The plaintiff was called in rebuttal and testified that she remembered when she was examined by this physician. She was then asked as to what he said about her condition, and answered,

"he told Dr. Robbins that this was the worst accident case he had handled in the last ten years." The defendant requested that the answer be stricken out; and the judge admitted it only so far as it tended to contradict any opinion or statement of the physician previously testified to by him. A majority of the Supreme Judicial Court is of the opinion that the evidence as so limited was admissible in the discretion of the presiding judge. The jury might have found that it was more or less inconsistent with the testimony of the physician on his direct examination, in that he believed the plaintiff's injury was more serious than he had testified it to be. The statement made in the hospital had a tendency to contradict his testimony, and for this reason might be found to affect its weight. As limited by the presiding judge, the substantial rights of the defendant were not injuriously affected.

In an Indiana case,[40] the Supreme Court decided that it was not reversible error to admit evidence that comparison of both knees by roentgen examinations of both knees of the plaintiff, to show that one knee had been injured.

I have repeatedly shown roentgenograms of a normal skull and at the same time exhibited the skull, when appearing as an expert in cases involving the question of skull fracture, when roentgenograms previously introduced in the case and purporting to show fractures were not such, but were the roentgenograms of skulls showing unusual grooves, lines, etc.

I have also several times exhibited roentgenograms of the spinal column and of the pelvis of normal individuals to show the similarity or the difference between these and the roentgenograms previously introduced in evidence; but at no time has there been any question raised as to the admissibility of any of these.

In a New Jersey case,[41] it was decided that a physician witness may examine roentgenograms to refresh his memory, even though the roentgenograms were not identified or introduced in evidence. The court said, "Anything that will refresh the recollection of a witness may be looked at by him for that purpose, although in itself it is not evidential."

In an Ohio case,[78] decided by the Supreme Court of that state, photographic prints made from the roentgenograms of the injured parts of the plaintiff in the case—where the original roentgenograms were already in evidence—were decided to be inadmissible because they were only copies of the roentgenograms and were therefore not the best evidence obtainable.

In a recent Washington case,[83] where the medical experts differed as to the extent of the injury, a roentgenogram of a knee which had been more severely injured, was introduced and allowed in evidence. The Supreme Court of Washington decided that, under the circumstances, the trial court committed no error in admitting this roentgenogram in evidence.

A concise and clear cut statement of what expert testimony should do seems apropos here and we will quote the Supreme Court of Oklahoma, when it said,[69]

> Expert testimony should serve the purpose of making clearer to the ordinary mind, facts that are obscure and understood only by scientific study, skill and experience, and such testimony should be definite and certain, and explanatory of the point under consideration.

In closing, I find that I have studied eighty-three decisions emanating from the highest courts of the following thirty states, namely: Albama, Arizona, Arkansas, Delaware, Idaho, Illinois, Indiana, Iowa, Kentucky, Maine, Massachusetts, Minnesota, Missouri, Montana, Nebraska, New Jersey, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Texas, Utah, Vermont, Washington, West Virginia, Wisconsin and the District of Columbia. There are doubtlessly many more leading and important cases not mentioned or listed; but we feel that enough have been reviewed and quoted from to justify the remarks made at the beginning of this paper, particularly regarding the diversity of opinions and apparent lack of appreciation by the courts and the legal profession of the importance of the *legal aspect of identification and interpretation of roentgenograms.*

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20th Annual Meeting: Atlantic City, N.J., June 10–11, 1935.

EDITORIAL

ADOLESCENT KYPHOSIS

THE SUBJECT of kyphosis should be of particular interest to roentgenologists for it is upon the evidence produced in the roentgenogram that the final diagnosis is arrived at. The usual types of kyphosis such as the rachitic round back in small children, the typical dorsum rotundum of senility and the kyphosis incident to a chronic ankylosing inflammatory condition of the spinal column are well known, but as Edelstein[1] says, "inadequate recognition has been given to that antero-posterior curvature of the spine which occurs in adolescence and which is a definite clinical and pathological entity." This type of spinal deformity occurs in otherwise apparently healthy young boys and occasionally in girls and has no relation, so far as is known, to Pott's disease. If the disease remains unarrested it results in a fixed deformity of the spinal column.

Scheuermann of Copenhagen was the first to give a full account of the condition and to give suggestions as to its etiology. He was the first to draw attention to the significance of the epiphyses above and below the bodies of the vertebrae and named the disease "osteochondritis deformans juvenilis dorsi," though the condition is usually referred to as Scheuermann's disease. He drew an analogy between the pathologic process observed in the vertebral column and the changes which occur in the upper epiphysis of the femur in Legg-Perthes' disease and at the tibial apophysis in Osgood-Schlatter's disease. Curiously enough, the disease occurs in adolescents of athletic build and they are usually endowed with a strong muscular and osseous system.

A knowledge of the disease is of importance because if it is recognized in its early stages it is amenable to treatment, but untreated it results in a fixed deformity of the spine leading to a typical dorsum rotundum seen in adult life, with the narrowed vertebral bodies which are often mistaken for compression fractures in the adult. The disease usually occurs during the active growth of the individual, usually between twelve and seventeen years, and is more frequent in the male; rarely does it occur in the female. The curvature is almost always in the dorsal region of the spine, though cases have been described with a lumbar spinal involvement. Clinically the back shows very little or no rigidity on active movement and the patient can move about freely, though in the region of the kyphos the back is completely fixed and usually cannot be actively or passively corrected. The kyphos is not sharp but gradually rounded and smooth, and, as a rule, is most prominent in the vicinity of the seventh to the tenth dorsal vertebra.

The involved vertebral bodies give a characteristic roentgen appearance, the roentgenographic appearance varying according to the stage of the disease process. For a proper interpretation of the changes observed in these vertebral bodies a knowledge of the normal ossification of the vertebra and its roentgenographic features is necessary. Under normal conditions the ossification of the vertebral bodies occurs from three bony centers: one in the body and two in the arches, which gradually increases until arches and body become united sometime between the third and eighth year of life. The subsequent growth of the vertebrae depends upon the periosteal bone formation. About the twelfth year of life there appears at the upper and lower anterior edges of the vertebral bodies a small tri-

[1] Edelstein, J. M. Adolescent kyphosis. *Brit. J. Surg.*, July, 1934, *22*, 119–133.

angular roentgen shadow representing the earliest signs of the epiphyseal ring. These were first described by Köhler. They are first observed in the lower dorsal region and then are subsequently seen extending up and down the column. These small shadows lie on the anterior superior and anterior inferior corners of the vertebral bodies. These ring-shaped formations gradually extend backwards thinning out in a fine line and assuming greater thickness again at the posterior edges of the vertebral bodies. This ring remains separated from the body of the vertebra until the time of fusion which, according to Schulthess, is about the twenty-fifth year, at which time the length growth of the vertebral bodies is concluded. It is only in the lateral view of the spine that these epiphyses are clearly seen. In adolescent kyphosis these vertebral body epiphyses may become rarefied, irregular and their outlines are somewhat frayed, and the adjacent vertebral bodies show indistinctness of outline above and below suggesting some abnormal process at the line of growth. The changes are more marked anteriorly and, as a result, the anterior surfaces of the vertebrae become shorter than the posterior and the wedge-shaped deformation of the bodies is often seen quite early with the intervertebral spaces no longer clear and the normal intervetebral disc spacing considerably diminished. If the disease process continues, actual fragmentation of the epiphyseal plates may occur. The process is usually limited to two, three or four vertebrae. The resulting deformities of the vertebral bodies of course vary with the severity of the process.

The exact etiological factor is undetermined but most observers agree with Scheuermann that it is a disturbance akin to Calvé-Legg-Perthes' and Köhler's disease, namely, a primary disturbance of the primary ossification process in the epiphyses, and the exact causative factor in the irritation of the epiphyses has not been determined. Steindler[2] says that "In default of better reasons, there seems no objection, then, to classify the condition with late rickets, or with other weight-bearing deformity of adolescence, such as genu varum, genu valgum, or coxa vara; but conclusive evidence could only be found by establishing chemical changes similar to those in manifest rickets." But Edelstein suggests that the etiology must be sought for in "some hitherto undetermined biochemical disturbance to explain why the epiphyses of some children fall victims to circumstances which in others have no effect." Whatever the cause it is of paramount importance to recognize it because it is amenable to orthopedic treatment, and since the final diagnosis rests in the roentgen examination, it is necessary for the roentgenologist to be familiar with the disease process and not to confuse it with the other forms of kyphosis.

In its active stage a differential diagnosis must be made between Pott's disease but the multiplicity of vertebral involvement and the characteristic appearance of the vertebral epiphyses should make the diagnosis easy.

[2] Steindler, Arthur. Diseases and Deformities of the Spine and Thorax. C. V. Mosby Co., St. Louis, 1929, pp. 114–125.

It is with regret that we announce the recent death of Dr. Pasquale Tandoja. Dr. Tandoja was a former president of the Societa Italiana di Radiologia Medica and at the time of his death was a member of the Executive Committee of the International Congress of Radiology and a member of the Editorial Board of *Archivio di radiologia*. His death is a great loss to radiology.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. E. P. Pendergrass, University Hospital, Philadelphia, Pa. Annual Meeting: Atlantic City, N. J., Sept. 24–27, 1935.

American College of Radiology
Secretary, Dr. Albert Soiland, 1407 S. Hope St., Los Angeles, Calif. Annual meeting: Atlantic City, N.J., 1935.

Section on Radiology, American Medical Association
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: Atlantic City, N.J., 1935.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N.Y. Annual meeting: Memphis, Tenn., Dec. 3–7, 1934.

Radiological Section, Baltimore Medical Society
Secretary, Dr. W. B. Firor, 1100 N. Charles St., Baltimore. Meets third Tuesday each month, September to May.

Radiological Section, Los Angeles County Medical Society
Secretary, E. N. Liljedahl, Los Angeles, Calif. Meets on the third Wednesday of each month at the California Hospital.

Radiological Section, Southern Medical Association
Secretary, Dr. F. B. Bogart, Medical Arts Bldg., Chattanooga, Tenn. Annual meeting: San Antonio, Texas, November, 1934.

Brooklyn Roentgen Ray Society
Secretary, Dr. A. L. Voltz, 115 Myrtle Ave., Richmond Hill, N.Y. Meets monthly on first Tuesday, October to April.

Buffalo Radiological Society
Secretary-Treasurer, Dr. Joseph S. Gian-Franceschi, 610 Niagara St., Buffalo, N.Y. Meets second Monday of each month except during summer months, place of meeting selected by the host.

Chicago Roentgen Society
Secretary, Dr. R. G. Willy, 1138 N. Leavitt. Meets second Thursday of each month October to May inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. H. G. Reineke, General Hospital, Cincinnati, Ohio. Meets third Monday of each month, October to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. Otto Glasser, Cleveland Clinic. Meetings are held at 6:30 P.M. at the Cleveland Chamber of Commerce Club rooms on the fourth Monday of each month from October to April, inclusive.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne Country Medical Society Building.

Florida Radiological Society
Secretary, Dr. W. McL. Shaw, 418 St. James Bldg., Jacksonville, Fla. Meetings held twice a year, May and November.

Illinois Radiological Society
Secretary, Dr. H. W. Grote, 219 N. Main St., Bloomington, Ill. Regular meetings held quarterly.

Indiana Roentgen Society
Secretary, Dr. J. N. Collins, Indianapolis, Ind. Annual meeting each February 22 in Indianapolis.

Michigan Association of Roentgenologists
Secretary, Dr. S. W. Donaldson, St. Joseph's Mercy Hospital, Ann Arbor, Mich. Three meetings a year, Fall, Winter, and Spring.

Milwaukee Roentgen Ray Society
Secretary, Dr. S. A. Morton, Columbia Hospital, Milwaukee, Wis. Meets monthly. Place of meeting designed by the president.

Minnesota Radiological Society
Secretary, Dr. L. G. Rigler, University Hospital, Minneapolis, Minn.

New England Roentgen Ray Society
Secretary, Dr. Thomas R. Healy, 370 Marlboro St., Boston Mass. Meets monthly on third Friday, Boston Medical Library.

Radiological Society of New Jersey
Secretary, Dr. P. S. Avery, New Brunswick, N. J. Meets annually at time and place of State Medical Society. Mid-year meetings at place designated by the president.

New York Roentgen Society
Secretary, Dr. E. F. Merrill, 30 W. 59th St., New York. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

North Carolina Roentgen Ray Society
Secretary, Dr. Major Fleming, Rocky Mount, N.C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

Central New York Roentgen Ray Society
Secretary, Dr. F. C. Rulison, Medical Arts Bldg., Syracuse, N.Y. Three meetings a year—January, May, and November.

Pacific Roentgen Club
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association

Pennsylvania Radiological Society
Secretary, Dr. L. E. Wurster, Williamsport, Pa. Annual two-day meeting held during May, 1935, meeting to be announced.

Philadelphia Roentgen Ray Society
Secretary, Dr. Thomas Loughery, Germantown Hospital. Meeting first Thursday of each month from October to May inclusive, at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

Rochester Roentgen Ray Society, Rochester, N.Y.
Secretary, Dr. W. W. Fray, Strong Memorial Hospital. Meets monthly on second Thursday from October to May, inclusive, at 7:45 at the Rochester Medical Association Building.

St. Louis Roentgen Ray Society
Secretary, Dr. P. C. Schnoebelen, Missouri Bldg., St. Louis, Mo. Meets first week of each month. Time and place of meetings designated by president.

South Carolina X-Ray Society
Secretary, Dr. R. B. Taft, 105 Rutledge Ave., Charleston. Meets at time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. F. B. Bogart, 311 Medical Arts Bldg.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary-Treasurer, Dr. J. H. Smith, Lubbock, Texas
Meets annually one day preceding the meeting of the Texas State Medical Association.

University of Michigan Department of Roentgenology Staff Meeting
Secretary, Dr. C. B. Peirce, University Hospital, Ann Arbor, Mich.
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Roentgen Club
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis.
Meets monthly on last Thursday, October to April, Service Memorial Institute, at 4:30 P.M.

Virginia Roentgen Ray Club
Secretary, Dr. V. W. Archer, University Hospital, University, Va.
Meets annually in October.

Cuba

Sociedad Cubana de Radiologia y Fisioterapia
Secretary, Dr. Francisco Padron, Enrique, Villuendas 64, Havana, Cuba. Meets monthly in Havana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Meets on the third Thursday of each month, from November to June inclusive, at 8:15 P.M., at 32 Welbeck St., London, W. 1, or as advertised.

Electro-Therapeutic Section of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month during the winter at 8:30 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Colin Macdonald, Lister House, 61 Collins St., Melbourne, Australia.
Meets monthly at Melbourne during the winter.

Section on Radiology, Canadian Medical Association
Secretary, Dr. A. H. Rolph, 160 St. George St., Toronto, Ont.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. P. C. Fenwick, The Hospital, Christchurch. Meets annually.

Continental Europe

Belgian Society of Roentgenology
Secretary, Dr. J. Boine, Avenue des Alliés, 134, Louvain (Belgium).
Meets monthly on second Sunday at d'Egmonds Palace, Brussels, except in the summertime.

Société de Radiologie Médicale de France
Meets monthly on second Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean, La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse, Biel.
Meets annually in different cities.

Société Francaise d'Electrothérapie et de Radiologie Médicale.
Meets monthly on fourth Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

Association of German Roentgenologists and Radiologists in Czecho-Slovakia
Secretary, Dr. Walter Altschul, German University, Prague, 11.52.

Deutsche Röntgen-Gesellschaft (Gesellschaft für Röntgenkunde und Strahlenforschung)
Meets annually in April, alternating one year in Berlin, one year in some other German city. Meets in addition every two years with the Gesellschaft deutscher Naturforscher und Aerzte.
Permanent secretary, Professor Dr. Haenisch, Klopstockstrasse 10, Hamburg, Germany.

Süd- und Westdeutsche Röntgengesellschaft
Meets annually in different cities.

Nord- und Ostdeutsche Röntgengesellschaft
Meets annually in different cities.

Dutch Society of Electrology and Roentgenology
Holds two meetings a year in Amsterdam, one in the Spring, and one in the Fall.

Societa Italiana Radiologia Medica
Secretary, Professor M. Ponzio, University of Turin, Turin.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Nicolae Busila, 44 Elizabeta Blvd., Bucarest.
Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad, USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson.
Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin.
Meets monthly, first Monday at 8 o'clock State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky.
Meets monthly on the first Monday at 8 o'clock, the place of meeting being selected by the Society.

Polish Society of Radiology
Secretary, Dr. Jan. Kochanowski, 45 Gornoslazka St., Warsaw. Meets annually.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. B. Krynski, 11 Zielna St.
Meets once a month except in the summertime.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association. Each of the following societies, with exception of the Denmark Society, meets every second month except in the summertime.

Society of Medical Radiology in Sweden
Meets in Stockholm.

Society of Medical Radiology in Norway
Meets in Oslo.

Society of Medical Radiology in Denmark
Secretary, Dr. G. Biering, Copenhagen.
Meets on the second Wednesday of each month from October to July in Copenhagen, at 8 o'clock in the State Institute of Roentgenology.

Society of Medical Radiology in Finland
Meets in Helsingfors.

Vienna Society of Roentgenology
Secretary, Professor Holzknecht, Vienna, IX, General Hospital.
Meets first Tuesday each month, October to July.

Orient

Japan X-Ray Association
c/o Orthopedic Surgery, Tokyo Imperial University.
Meets annually in April.

Kinki Roentgen-Abend Society
Director, Dr. Prof. Taiga Saito, Ogawaoike Tyoto, Japan. Meets bi-monthly on third Sunday.

DANGERS OF ROENTGENOSCOPY

To the Editor:

The paper under the heading of "The Dangers of Roentgenoscopy and Methods of Protection against Them" by Drs. Leddy, Cilley and Kirklin* is of very great practical importance, for it is based on biological experience and an attempt to find the via media between over-caution and cramped efficiency, on the one hand, and risk of damage to the observer, on the other. The authors quote my work in collaboration with Sydney Cox† in which we attempted to estimate the dangers and to show what degree of protection was necessary. The problem had hitherto been regarded as a physical one and the figures of Glocker and Mutscheller were entirely empirical and based solely on physical data. Yet the problem is primarily a biological one and must be solved by the roentgenologist from his experience. He only can define the conditions of danger and it is at this point that the physicist enters the field to measure the intensities of radiations that the radiologist has used in his experiments. Working from a computation of large daily doses that were known to have been tolerated without ill effect for seven years, we suggested a very conservative estimate of the daily dose of radiation that was safe. This was at a time when the pendulum had swung to excessive caution and the majority of roentgenologists interested in the subject inclined to the view that *absolute* protection was essential—we were not sure whether or not small doses were dangerous. Our work stated a fact that all practical roentgenologists knew, i.e., that small doses produced no observable ill effect and were therefore, presumably, safe. The safety limit that we suggested was 1/25 of what had been known to be tolerated without ill effect (i.e. 0.00028 U.S.D., not 0.0028 U.S.D. as erroneously stated in the paper under discussion). Of set purpose we knowingly erred on the over-cautious side to avoid criticism and in the hope that our obvious over-cautiousness would influence the more timorous. The British X-ray and Radium Protection Committee was revising its recommendations at that time and it was hoped that this attempt to find a trustworthy biological safety dose would counteract the tendency to the demand for absolute protection that was cramping roentgenoscopy by making apparatus expensive and unwieldy by reason of the excessive degree of protection prescribed.

Subsequent observations, that are so strongly supported in this new contribution, strengthened the impression that our safety limit of 0.00028 U.S.D. was lower than necessary. Moreover, the accurate measurements of the intensities involved have confirmed us in our beliefs. The photographic methods of measurement we used in 1926 were unsatisfactory as they allowed too wide a margin of error. Ordinary iontoquantimeter readings allow of only one observation at a time and the instruments are not usually calibrated to small intensities. Moreover, it is not always easy to apply the ionization chamber at the spot where we wish to measure the intensities. The ideal method for this purpose is the use of small condenser chambers of the Sievert type ($\frac{1}{2}\times\frac{1}{4}$ inch), which are placed in the position in which readings are desired. These are all exposed at the same time and the readings made at once. W. V. Mayneord very kindly brought his apparatus to my laboratory and carried out the measurements with the ionization chambers and instruments he has designed. The readings were very satisfactory and gave accurate information.

The conditions of screening were reproduced and a cadaver was used for the observations. In this way we measured the intensities of radiation sustained by the hand in palpation and by the abdomen of the observer. A paper based on these data was read at the Fourth International

* Am. J. Roentgenol. & Rad. Therapy, Sept., 1934, *32*, 360–368.

† The radiation risks of the roentgenologist. Am. J. Roentgenol. & Rad. Therapy, 1928, *19*, 551–561.

Congress of Radiology held at Zurich and will be published shortly. In brief, I suggested that our 1928 figure (0.00028 U.S.D. or 0.188 r) was unnecessarily conservative and from experience came to the conclusion that ⅛ instead of 1/25 of the known tolerated dose, i.e., a daily dose of 0.0014 U.S.D. (0.94 r) was still well within the safety limits.

We found that the dose received by the palpating hand in the direct beam was 0.2 r for each five minutes' exposure under the conditions of ordinary screening, assuming that the hand was in the direct beam the whole time, as of course it is not in actual practice. In twenty-five minutes, therefore, the daily safety limit of 0.94 r is exceeded, i.e., I came to the same conclusion as the writers of this article—palpating in the direct beam gives an exposure on the hand that falls very near the border line of safety. Hence the expert who screens rapidly, "hides" his hand behind the barium shadow and uses a small diaphragm, is probably safe, but that the inexpert worker who does not use a skillful and rapid technique is in danger. I quite agree that washleather or other leather gloves are of no value as protection and that the only way of safeguarding is either to keep the hand out of the direct beam or to use a protective glove. Such a glove, however, need not have more than a fractional lead equivalent since as little as ⅛ mm. lead will cut down this type of radiation to about 2 per cent. It would be easy to work out the lead equivalent that would reduce only to 50 per cent or even 25 per cent and this would, I believe, give an ample margin of safety, and a protective glove of this value could be made so thin and pliable that it would not seriously interfere with manipulation or vision.

Dr. Carman and I discussed the matter several times and it was as the result of our conversations that we both abandoned the use of protective gloves, holding that safety lay in speed and skill in palpation and that the use of gloves limited the facility and speed of the examination, interfered with the field of vision and gave a sense of false security.

However, I never felt certain that my hand was safe in the direct beam and therefore devised a technique for palpating from just outside the illuminated area.* Measuring the intensities of radiations to which my hand is exposed with this technique, the readings gave 0.01 r for five minutes' exposure. Clearly then the hand palpating just outside the direct beam is perfectly safe.

The outstanding deduction from our measurements was the definite danger to the observer standing beside the couch, particularly with the wide open diaphragm we use for most of the barium enema work. In these conditions 0.8 r was measured in five minutes. Practically the whole of this was secondary radiation coming from the subject, and would strike the observer's abdomen. The clothes give only a very small degree of protection and one would have expected to hear more often of damage to the skin of the abdomen and perhaps of intestinal disturbances in those who have not recognized the danger and have not used an apron. As already stated, ⅛ mm. lead cuts this type of radiation down to 2 per cent and is therefore ample to produce conditions of safety, i.e., there is no need whatever for the massive aprons of 2 or 3 mm. lead equivalent that usually adorn the walls of roentgen departments. A light apron *that is used* is all that is necessary—a thick apron that is too cumbersome to wear and hangs stiff on its peg shows that the roentgenologist has not studied the problems and is ignoring a danger that is definite. Those who use these heavy aprons add materially and unnecessarily to the fatigue of work that is exacting and for which all reserves of energy are required.

In conclusion, may I express my appreciation of this excellent and practical attempt to lay the bogey of the dangers of screen examinations.

A. E. Barclay

The Medical Schools
Cambridge, England

* Barclay, A. E. The dangerous art of palpation. *Brit. J. Radiol.*, 1926, *31*, 385–393.

MEMORIAL TO DR. STANLEY MELVILLE

The following communication will be of interest to the many friends of the late Dr. Stanley Melville:

TO THE EDITOR OF THE TIMES

Sir,—The dangers of X-ray work were brought home vividly to the medical world by the death of several of our pioneer radiologists. In 1921 a letter to *The Times* announced the formation of a special committee to advise on these dangers. In this action the late Dr. Stanley Melville played a very prominent part.

This "X-Ray and Radium Protection Committee" was the first in the world to draw up recommendations for safeguarding those who work with these agents. Their value may be gauged by the fact that they formed the basis of the recommendations adopted at the Stockholm International Radiological Congress in 1928.

Dr. Stanley Melville took a most important but unobtrusive part in all that concerned medical radiology. He was one of those who worked for the establishment of the Diploma in Medical Radiology and Electrology (D.M.R.E.) at Cambridge in 1919, and subsequently the teaching and examinations connected with it were his constant care. The bi-lingual Congress of 1922 and the First International Congress of Radiology in London in 1925 were largely due to his initiative, as was also the formation of the British Institute of Radiology with which the Röntgen Society was eventually incorporated. He did much for the welfare of the lay worker and took a prominent part in founding the Society of Radiographers. Sadly handicapped physically and often suffering, as the result of dermatitis incurred in the early days, Melville never spared himself when any work for medical radiology had to be done; that he should be ever ready to step into the breach seemed to be the natural course of events.

Some of your readers may feel that his services should be recognized and perpetuated by a suitable memorial, such as a travelling fellowship in radiology. Donations, however small, for the purpose will be gratefully acknowledged. At a later date a meeting of those subscribing to the fund will be called in order to decide what form the memorial shall take and how he funds subscribed shall be administered.

We are, yours faithfully,

HUMPHRY ROLLESTON (Chairman).
RUTHERFORD (Cambridge).
L. S. T. BURRELL (Brompton Hospital).
G. W. C. KAYE (National Physical Laboratory).
G. W. MITCHELL (St. George's Hospital).
R. S. PATERSON (Radiology Section, Royal Society of Medicine).
LEO A. ROWDEN (Society of Radiographers).
SIDNEY RUSS (British X-Ray and Radium Protection Committee).
J. DUNCAN WHITE (British Institute of Radiology). } Hon.
A. E. BARCLAY (British Institute of Radiology) } Secretaries

32, Welbeck Street, W.1.

London Times, Aug. 2, 1934.

NOTICE

Dr. Jenkinson, the treasurer of the American Roentgen Ray Society, requests that the radiologists to whom he sent questionnaires regarding the Physicians and Surgeons Liability Insurance return these to him at once properly filled out. He is anxious to get this information in tabular form to present to the various insurance companies.

A CORRECTION

In the article on "The Migratory Cecum" by Jacob Buckstein in the August, 1934, issue of the Journal, the last sentence in the first paragraph on page 173 reads as follows: "Thus, in 91 per cent the lowest point of the cecum was below the interiliac line (45.6 per cent of cases)."

The sentence should read as follows: "Thus in 91 per cent the lowest point of the cecum was below the iliac fossa, and its most common position 5 to 6 inches below the interiliac line (45.8 per cent of cases)."

DEPARTMENT OF TECHNIQUE

Department Editor: ROBERT B. TAFT, M.D., Ph. D., 105 Rutledge Ave., Charleston, S. C.

THE IMPORTANCE OF MEASURING r OUTPUT OF ROENTGENOSCOPIC EQUIPMENT

By LAWRENCE M. HILT, M.D.
SPRINGFIELD, ILLINOIS

MOST roentgenoscopic equipment now in use antedates both the international r and the general usage of ionization measurements. Different types of material are interposed between the tube and patient ranging from cardboard and bakelite to 0.5 mm. Al. Most machines do not have the aluminum filter.

Five minutes is usually assumed to be a safe time at the usual roentgenoscopic setting and distance. However, as has been shown before, the output of different machines and tubes, especially at low kilovoltage, is an exceedingly variable factor.

Having three different roentgenoscopes, two of which operate off the same transformer, it seemed worth while to have these calibrated for r output per minute, as installed, and then to add various thicknesses of aluminum filter. The calibration was done by R. S. Landauer with the following results:

Roentgenoscope A.
As installed by the manufacturer
r output per minute 40
Safe dose 250 r
Safe time 6¼ minutes
1 mm. Al filter used
r output per minute 17.7
Erythema dose 250 r
Safe time 14.1 minutes

Roentgenoscope B.
As installed by the manufacturer
r output per minute 30
Safe time 8.3 minutes
1 mm. Al
r output per minute 14
Safe time 18 minutes
2 mm. Al
r output per minute
Safe time 25 minutes

Roentgenoscope C.
As installed by the manufacturer
r output per minute 23
Safe time 11 minutes
1 mm. Al
r output per minute 13
Safe time 19 minutes

Roentgenoscopes B and C operate from the same transformer.

It is interesting to note the variance from 40 r per minute to 23 r per minute. With the addition of 1 mm. Al filter it will be noted that the output of all three tubes is brought more nearly to the same level. In my experience 1 mm. Al does not interfere with the detail in a roentgenoscopic examination; 2 mm. Al interferes slightly with the detail but gives adequate visibility for reducing fractures of the long bones when it is desired to do this under the roentgenoscope.

It is true that all these machines were within the five minute safety limit, but there is almost twice the safety factor of Roentgenoscope C compared with Roentgenoscope A.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

NECK AND CHEST

CAPUA, ANTONIO. Aspetti radiologici dell'oleotorace. (Roentgen appearance of oleothorax.) *Radiol. med.*, Sept., 1933, *20*, 1194–1200.

The author describes the roentgen appearance of the chest in which oleothorax has been used and illustrates it with ten roentgenograms. The accumulation of oil shows a homogeneous shadow of slight density, not much greater than that of soft parts. Its density decreases from below upward and at the apex there is very little difference from the opposite side, even in complete oleothorax. When the oil is used to prevent adhesions it tends to assume the shape of a large ovoid with an oblique longitudinal axis. Its outer border is at the wall of the thorax and its inner edge at the boundary between the oil and the compressed lung. When the oil is used for disinfectant purposes it takes on the form of the exudate which it has replaced. The ribs are distinctly visible in the oil shadow while they are not visible in the dense shadow of an exudate. There are sometimes dense lines in the shadow which represent adhesions. Sometimes a reactive exudate forms in the oleothorax sac which because of its greater specific gravity collects in the lower part with the oil above it. There is a level between the two liquids which follows hydrostatic laws. Sometimes on the inner convex margin of the oil shadow there is a denser band due to visceral pleurisy or to atelectasis of the peripheral layers of the lung. The oil in the thorax acts like a foreign body and sometimes causes a plastic reaction of the folds of the pleura limited to the area with which the oil is in contact which produces a shadow of greater intensity than that of the oil.—*Audrey G. Morgan.*

GRANDGÉRARD, ROGER, and WÉBER, PIERRE. Les plans scissuraux du poumon et leurs rapports médiastino-diaphragmatiques; étude anatomo-radiologiqua. (The scissural surfaces of the lung and their mediastinal diaphragmatic relation; anatomic roentgenologic study.) *J. de radiol. et d'électrol.*, Dec., 1933, *17*, 649–655.

Since the comprehensive work of Luschka in 1857, numerous articles have been published on the pulmonary scissuras dealing, in the beginning, with the anatomy and external topography of the interlobes and, after the introduction of the roentgen investigation, with the appearance of the borders of the various hidden portions of the lungs and their best mode of projection on the screen or film. It is interesting, however, that none of the investigators have studied the precise mediastinal and diaphragmatic relationship of the scissuras. For this reason the authors undertook the task to analyze the pulmonary scissuras under their true aspect, that is, in describing them as surfaces without consideration of the frequent change from the normal and by entirely omitting the study of their angulation with the horizontal plane which has been so well put to the point by D'Hour. They also studied the mediastinal diaphragmatic relation of the scissural leaves. The material used included lungs of cadavers fixed according to the typical anatomic procedure, from which plaster-of-Paris models were then made. The work was a painstaking one but it has permitted the precise evaluation of the configuration of the scissural surfaces and especially their mediastinal diaphragmatic relations. These are described in detail in the article and illustrated with several schematic drawings and a few topographic projections of the scissuras which are important from the point of view of roentgen examination of the chest.—*T. Leucutia.*

JALET, J. Étude radiologique du lobe cardiaque, et de la scissure supplémentaire qu'il engendre; lobite et scissurite cardiaque. (Roentgen study of the cardiac lobe and of the supplementary scissura which delimits it; lobitis and cardiac scissuritis.) *J. de radiol. et d'électrol.*, Dec., 1933, *17*, 656–662.

The cardiac or infracardiac lobe which was first erroneously called an "azygos lobe" constitutes a true lobe of the lung including the territory of a primary bronchus, the so-called infracardiac bronchus. The anomaly is interesting from the standpoint of the roentgenologist since it occurs rather frequently. The author in collaboration with Bourdellés and Amigues found it in 17 cases out of 170 fetal lungs which is a figure considerably smaller

than indicated by the statistics in most of the other publications. Roentgenologically the normal cardiac scissura is difficult to detect, its shadow as a rule becoming lost among other shadows of the normal bronchovascular tree. According to Ottonello it appears as a straight or peripherically convex thin capillary line which starts at the vault of the diaphragmatic dome, continues upward and inward and becomes lost in the right hilar region. Its exact course is extremely variable, the starting point appearing any place within the internal two-thirds of the right diaphragmatic dome and its upper termination reaching different points either below or above the horizontal scissura. The image is never observed on the left side where it is overlapped by the cardiohilar shadow.

The true importance of a cardiac lobe becomes manifest in pathological conditions. After an analysis of another series of more than 3,000 adult roentgenograms, the author found several in which the diaphragmatic hilar linear shadow was well demonstrated. The majority of these were from cases which clinically were convalescents from serofibrinous pleuritis, showing all the signs of pachypleuritis of the right base. The diffierential diagnosis includes the triangular shadow of mediastinal pleurisy, the images of paramediastinal bronchiectasis, retrocardiac aneurysm, cold abscess of vertebral origin and esophageal dilatation.

The conclusion is reached that the cardiac lobe merits attention in the differentiation from opacities or other images of the right base. —*T. Leucutia.*

Ledoux-Lebard, R., and Garcia-Calderon, J. Les lobes accessoires de la base pulmonaire (lobes cardiaques). (Accessory lobes of the pulmonary base: cardiac lobes.) *Bull. et mém. Soc. de radiol. méd. de France*, Nov., 1933, *21*, 718–722.

Numerous articles have appeared recently in the literature dealing with the azygos lobe but the other accessory or supernumerary lobes of the lung have received little attention. In considering the accessory lobe of the base, the so-called cardiac lobe, the authors found that the condition is quite common, a roentgen demonstration being possible in 5 per cent of all the cases (anatomists claim that the cardiac lobe occurs in 25 to 30 per cent of cases). The chief roentgen characteristics are: a linear shadow to the right of the heart which is, as a rule, convex toward the outside; a slight flattening of the dome of the diaphragm as if the cardiac lobe has left an imprint on it, and, finally, an indentation at the point where the linear shadow merges with the diaphragmatic dome.

The importance of the cardiac lobe becomes evident in case of various lesions localized to this area such as acute pneumonia or bronchopneumonia, tuberculous lobitis and scissuritis, bronchial dilatations, etc.—*T. Leucutia.*

Costedoat, A. La lymphangite cancéreuse des poumons à forme suffocante. (Suffocating form of cancerous lymphangitis of the lungs.) *Presse méd.*, May 10, 1933, *41*, 745–748.

The suffocating form of cancerous lymphangitis of the lungs is an affection, the clinical symptoms of which are analogous to those of miliary tuberculosis invading the lungs. There are only a few articles published in the literature on the subject and therefore the author on the basis of one of his own cases wishes to consider the essential clinical features of this condition. He also reviews the few reported cases and by coordinating all that has been described he now tries to establish the disease as a distinct clinical entity. Briefly, the following are the characteristics: The condition generally occurs in young subjects between the ages of thirty-five and forty, and nearly always represents a metastatic process from a cancer of the stomach or very rarely from a cancer of the breast. The chief clinical symptoms are the respiratory disorders consisting essentially in dyspnea which is moderate in the beginning but rapidly increases in intensity, and spells of a very marked dry hacking cough which occasionally leads to suffocation. The pulse is very rapid, from 120 to 160, the arterial tension low, and there is a slight fever as high as 38°C. The roentgen examination furnishes important information revealing that the pulmonary lesions are more severe than could be suspected from the stethoscopic examination. The pulmonary parenchyma appears diffusely fogged as if covered with a dense and continuous lacy network. Where the linear shadows of the network cross each other, more opaque patches appear suggestive of tuberculous granules. Death usually occurs by dyspneic paroxysms.

The differential diagnosis includes the usual cancerous metastases to the lungs, the so-called massive embolic form of carcinomatous metastases and especially the acute tuberculous asphyxia of Graves. A differentiation from this

latter often becomes impossible except perhaps for the fact that the temperature is more elevated in the case of tuberculosis. It is concluded that the suffocating form of secondary cancer of the lungs presents a double interest, first, because it shows that the entire clinical history, symptoms and evolution, of acute tuberculous asphyxia can be produced by a cancerous process, and secondly because it suggests by analogy that an invasion of the lungs via the lymphatic path (and not the blood stream) is possible also in tuberculous infection from a primary focus of the mesenteric glands.—*T. Leucutia.*

Carey, J. B. Pneumosilicosis and amyloidosis. *Ann. Int. Med.*, Feb., 1933, *6*, 1106–1113.

A case with the unusual association of pulmonary silicosis and renal and splenic amyloidosis is reported. Death was due to uremia by progressive renal impairment from the amyloidosis. The association of the two entities is considered merely a coincidence rather than an explicable relationship. A complete necropsy report with chest roentgenogram and photograph of the sectioned lung is presented.—*W. R. Brooksher.*

Reimann, S. P., and Seabold, P. S. Correlation of x-ray picture with histology in certain breast lesions. *Am. J. Cancer*, Jan., 1933, *17*, 34–41.

Roentgenograms of female breasts were taken at intervals before, during, and after menstruation and pregnancy. Fluctuations in size of the breast tissue proper were found corresponding to these physiological states. Two typical breast lesions were also studied in relation to the menstrual cycle, and were found to fluctuate in size with the stages of this process. These were periductal fibromas and abnormal involution. The lesions were removed surgically and their histology was found to correspond to the theoretical condition for the time of removal in relation to menstruation. It is not recommended that removal of doubtful lesions or of malignant tumors be delayed in order to take a series of roentgenograms. Attention to the menstrual phases and their different pictures is necessary, however, for proper pathological diagnosis of breast lesions.—*J. R. Andrews.*

ABDOMEN

Knutsson, Folke. On limy bile. *Acta radiol.*, 1933, *14*, 453–462.

The author reports 12 cases of what he calls limy bile, all of which were operated on. The gallbladder in these cases contained an amorphous calcium precipitate consisting chiefly of calcium carbonate. The mass of calcium may be liquid, semisolid or even solid, and may vary from whitish to dark brown in color. Volkmann called it lime milk bile but this name is not appropriate because it is not always liquid; Phemister called it calcium carbonate stone but this name can be correctly applied only to the cases in which the mass is solid. The author thinks his term is a more correct designation for all cases as it does not tell anything about the color or consistency of the gallbladder contents but only emphasizes the abnormal lime content. The limy bile causes a very dense gallbladder shadow. Confusion with normal contrast filling in cholecystography may be avoided by taking films in the upright position showing sedimentation of the limy bile and by taking films after a fatty meal. As a rule, the gallbladder also contains a hydropic clear bile in which the calcium sinks to the bottom of the gallbladder. In roentgenograms taken in an upright position the limy bile shows a horizontal upper boundary.

In all the above cases there were cholesterin stones, most of which appeared as filling defects in the limy bile. In 9 of the cases the cystic duct was occluded by an impacted calculus. On microscopic examination the gallbladder wall showed chronic inflammatory changes and hypertrophy of the musculature.

Phemister assumes that the calcium is secreted through the gallbladder wall but the author does not believe this is true; he thinks it more likely that the calcium is precipitated from the bile because of the valvular closure of the cystic duct which interferes with the outflow of the bile but not with its inflow.—*Audrey G. Morgan.*

Niculescu, Petre, and Banu, N. Documente radiologice asupra patogeniei colicei hepatice. (Roentgenologic documents on the pathogenesis of hepatic colics.) *Bul. Soc. de radiol. si electrol. med. din România*, June, 1933, *2*, No. 6, 5–10.

The authors were fortunate in observing in 2 cases the appearance of the gallbladder after cholecystography during the actual attack of colic. In none of the cases was the fat meal administered so as not to influence the physiologic emptying of the gallbladder.

It was observed that the gallbladder contracted during the period of pain, thus confirming by documentary means the long suspected mechanism of hepatic colic. The beneficial effect of morphine in all cases of hepatic colic is due, therefore, not only to a general analgesic action but chiefly to the suppression of the painful contraction of the gallbladder.—*T. Leucutia.*

Vlădescu, Violeta. Kist hidatic al ficatului diagnolticat prin hepato-lienografie. (Hydatid cyst of the liver diagnosed by hepatolienography.) *Bul. Soc. de radiol. si electrol. med. din România*, April, 1933, *2*, No. 4, 18–20.

Hepatolienography was successfully used in the diagnosis of a hydatid cyst of the liver in a patient fifty-one years of age. Twenty-five c.c. of thorotrast was injected intravenously on two successive days, the total dose thus being 50 c.c. No untoward effects were observed following the injection. A roentgenogram made forty-eight hours later revealed an opacification of the spleen and liver. In the middle of the opacified liver shadow a clearer rounded zone the size of a large pineapple was observed which was easily diagnosed as a hydatid cyst. The findings were confirmed at operation.—*T. Leucutia.*

Nemours-Auguste. Étude de l'évacuation réflexe de la vésicule biliaire. (Study of the reflex evacuation of the gallbladder.) *Presse méd.*, July 12, 1933, *41*, 1106–1109.

For several years the author was surprised to see the variation in form and especially the rapidity of the evacuation of the gallbladder following the administration of a Boyden meal in cholecystography. He suspected that the emptying of the gallbladder is brought about not only by the duodenal passage of the meal but also by some other cause. To confirm this, he mixed a tablespoonful of an opaque salt with the Boyden meal and studied the passage of this through the gastrointestinal tract simultaneously with the emptying of the gallbladder. It was observed that the emptying of the gallbladder started before than the Boyden meal reached the duodenum. From this he concludes that there must be a bucco-vesicular reflex comparable to the reflex of Pavlov which determines, at least partly, the emptying of the gallbladder. The presence of food in the stomach enforces the action of this reflex without the necessity of alimentary passage through the duodenum.—*T. Leucutia.*

Martinotti, Giuseppe. Ulteriori osservazioni sui quadri radiologici corrispondenti a fasi di sviluppo dell'ulcera duodenale. (Recent observations on the roentgen images of the different phases of development of duodenal ulcer.) *Radiol. med.*, 1933, *20*, 1279–1301.

The author describes and illustrates with roentgenograms three typical stages of duodenal ulcer. In the first stage there is a large niche with an inflammatory halo around it. If it is taken in profile or obliquely it is a Haudek's niche. In the second stage the niche is small or possibly not visible at all and a constriction of the cavity of the bulb has taken place, dividing it into two sacs. In the third stage the cavity of the bulb has become transformed into a rigid or semi-rigid tube with a diverticular sac at the base. The different stages are illustrated with serial roentgenograms. Atypical pictures may be caused by perivisceritis.

The author does not agree with Gutmann's opinion that the bulb should always be only slightly filled for the demonstration of duodenal ulcer. While scanty filling may be desirable for demonstrating a niche it is not desirable for demonstrating deformities of the bulb and may even lead to mistakes, suggesting a narrow lumen when there are dilatations and diverticula that are not filled.

These facts limit the value of Gutmann's classification. To demonstrate all the different stages of the disease, particularly the third stage, the bulb should be filled with contrast medium. And Gutmann's N-shaped image, it is claimed, is not a sign of periduodenitis but is caused by insufficient filling of the bulb. Haudek's niche is not pathognomonic of callous ulcer. The shape of this niche does not depend on callosity of the margins and base of the ulcer but on its location on the margin of the bulb and the shape of its outline seen in profile.—*Audrey G. Morgan.*

Vita, Giulio. Genesi e significato dell'immagine radiologica nell'ulcera gastrica e duodenale. (Genesis and significance of the roentgen picture in ulcer of the stomach and duodenum.) *Radiol. med.*, July, 1933, *20*, 878–888.

Patients often come for examination with symptoms of ulcer of the stomach or duodenum and even with the roentgen picture of a niche but on operation no ulcer is found. The symptoms in such cases are generally attributed to reflex nervous disturbances but from examina-

tion of many such cases that have been operated on the author does not believe that this is true. The roentgenogram of a niche in relief is caused by very complex factors. It may be produced by an ulcer cavity, edema or more particularly by loss of tonus in the zone affected by the inflammation. Some cases that have shown a roentgenogram of ulcer in relief on operation show only a stellate scar of the serous membrane which is not elevated at all. Others show the mucous membrane intact but disorganization of the muscle bundles. Almost always there is a true anatomical explanation of the roentgenogram.—*Audrey G. Morgan.*

Morton, Charles B. Syphilis of the stomach. *Arch. Surg.*, Nov., 1932, *25*, 880–889.

Although the clinical diagnosis of syphilis of the stomach is made frequently and such cases are reported in large numbers, histologically proved instances of the disease are rare. Hartwell, in 1925, reviewed more than 200 cases that had been recorded and he concluded that in all but 27 of these the evidence presented was insufficient to prove the diagnosis. He stated 5 clinical features that, when found in combination, had been considered more or less diagnostic: (1) positive Wassermann reaction; (2) a gastric anacidity or markedly reduced hydrochloric acid content; (3) marked deformity of the stomach roentgenologically, particularly of the "dumb-bell" type, or with an abrupt margin between the healthy and diseased portions of the stomach; (4) less marked cachexia, emaciation, and anemia than would be found with such roentgen changes due to lesions other than those of syphilis; (5) improvement under antisyphilitic treatment. He feels, however, that "there are no characteristic subjective symptoms or objective findings upon which a diagnosis of syphilis of the stomach may soundly rest."

The disease is initiated in the stomach by the formation of a gumma in the submucosa and extends from there into the other coats. Through the breaking down of the gumma, particularly because of the action of the gastric juice, ulcers and ultimately scars may form; then only by the finding of other gummas or the remains of gummatous tissue can the syphilitic nature be established.

The case of a white man, aged forty-eight, complaining of "stomach trouble" of eight months' duration is reported. The Wassermann reaction of the blood was strongly positive on several occasions. Roentgen examination of the stomach showed a canalized lumen between the cardiac and pyloric regions. A six hour gastric residue estimated at 50 per cent was still present. A diagnosis of carcinoma of the stomach with obstruction was made. The lower two-thirds of the stomach was resected. Examination of the resected portion showed ulceration and scarring of the mucous membrane; round cell, plasma cell, and polymorphonuclear infiltration of the mucosa; fibrosis of the submucosal layer with cellular infiltration; obliterative endarteritis; and hyperplasia of the lymph follicles. These changes were considered proof that the disease was true syphilis of the stomach. The search for spirochetes was unsuccessful because, the author thinks, of the fact that the patient was given two doses of arsphenamine prior to operation.—*J. R. Andrews.*

Ledoux-Lebard, R., and Garcia-Calderon, J., Les techniques d'examen de la muqueuse du gros intestin. (Techniques of examination of the mucosa of the large intestine.) *J. de radiol. et d'électrol.*, Aug., 1933, *17*, 429–466.

This article represents the first part of the report presented at the First Annual Reunion of French Electroradiologists (Paris, October 12–14, 1933) and deals comprehensively with the following factors of the roentgen examination of the mucosa of the large intestine: (I) history; (II) principle of the method, (1) anatomic data, (2) physicochemical basis; (III) technique; (IV) various forms of opacification, (1) oral method, (2) rectal method, (*a*) opaque enema, (*b*) evacuation of the opaque enema, (*c*) insufflation, and (V) the value and difficulties of the method.

The following general conclusions are reached: The thin layer examination in the form used today represents a considerable progress in intestinal roentgen diagnosis. Yet the method is far from having enjoyed an enthusiastic reception from the specialists. This must be attributed to the real difficulties of application and to the delicate interpretation of the images obtained. To become of more practical value the method must be made simpler, more rapid, less expensive and more constant results must be obtained than at the present time. A very extensive bibliography is appended.—*T. Leucutia.*

GILBERT, R., and KADRNKA, S. Résultats cliniques de l'exploration radiologique du relief interne du gros intestin. (Clinical results of roentgen exploration of the internal relief of the large intestine.) *J. de radiol. et d'électrol.*, Aug., 1933, *17*, 401–427.

This represents the second part of the report on the examination of the mucosa of the large intestine presented at the same meeting (see above abstract) and deals chiefly with the clinical results. Here the following factors are being considered more in detail: (I) modifications of the relief images of functional origin; (II) modifications of the relief images due to extrinsic affections; (III) alterations of relief images due to primary lesions of the bowel (A) inflammatory lesions, (B) neoplasms.

The following general conclusions are reached: The roentgen examination of the internal relief of the large intestine includes the study of several elements such as: (1) the surface structure of the mucosa with its various forms of folds; (2) the continuity of the surface structure; (3) the state of the secretory layer (degree of viscosity or fluidity, membranes, etc.); (4) the modifications of the relief due to motor function, as, for example, the autonomous motor function and the extrinsic effect (contractions, respiratory movements of the patient, etc.); (5) the factor of constancy (connected with the anatomic structure, tonicity and turgescence): the degree of softness or hardness and the degree of flexibility or rigidity.

The phenomena resulting from these elements appear on the roentgen image in the following manner: (1) The surface structure of the relief: in the form of moulage or impregnation of the mucosa, the folds corresponding to the hollows of the opacified relief. (2) The modifications of continuity: in the form of images of addition or subtraction, etc. (3) The state of the secretory layer: this is characterized by the degree of impregnation which is a function of the modifications of the adhesive power. (4) The modifications associated with movement: these are detected by serial images and by palpation. (5) The constancy: this is studied by analyzing the volume of the folds, the transmitted movements and by insufflation.

The morphologic variations in the normal consist of (*a*) modifications of the internal relief due to differences of the digestive activity according to age (turgescence, development of the folds); (*b*) modifications of the relief due to factors which influence the hydric state and the neuromuscular tonus: (α) vascular or lymphatic stasis (hepatosplenic, cardiac, adenomesenteric affections, etc.), anemias, cachexias, etc.; (β) pharmacodynamic influences (pilocarpine, morphine, etc.), physical influences (cold), neuroglandular influences (Basedows's disease, etc.)

In addition, in the pathologic conditions the following modifications may occur: (*c*) modifications of the internal relief secondary to extrinsic abdominal affections (compressions, contractions, etc.), manifesting themselves as modifications of motility, consistency and structure; (*d*) modifications of the relief due to inflammatory conditions of the large intestine manifesting themselves chiefly as modifications of surface structure, secretory layer, impregnation and consistency of the mucosa; (*e*) modifications of the relief due to primary neoplasms of the colon manifesting themselves in breaking of continuity or in variation of consistency and of secretory layer; (*f*) modifications of the relief due to diverticulosis leading to the more or less known typical images; (*g*) in case of more deep-seated lesions such as inflammatory stenosis, or differentiation between malignant and benign tumors, a moderate insufflation associated with the above method may lead to very valuable information; (*h*) the moderate insufflation also helps in a study of the disturbances of secretory layer by further characterizing the type of irregularity (colitis).

All in all, the authors express the opinion that the criticisms so often heard are unjustified. Thanks to the technical improvement and especially to the experience acquired, the relief method forms not only a valuable supplement to the classical methods of roentgen examination but it permits the establishment of new criteria and signs which are important both from the physiological and pathological standpoint. For this reason the method should occupy a prominent place in diagnosis. The article is very richly illustrated with some rather unusual roentgenograms.—*T. Leucutia.*

SKELETAL SYSTEM

THOMA, K. H. A case of progressive atrophy of the facial bones with complete atrophy of the mandible. *J. Bone & Joint Surg.*, April, 1933, *15*, 494–501.

Thoma reports this case because, as far as he could ascertain, it is the only case on record

of complete atrophy of a bone, and because its etiology presents probably an unanswerable question. The disease was initiated by dental infection. This infection was completely cleared up by extraction of the teeth and subsequent treatment. During a long interval the patient's jaw was normal and dentures were worn with comfort. At the end of her third pregnancy, pain started to appear in her jaws, which was the first indication of the disease. The pathological process at that time apparently caused fragility of the bone, which predisposed to pathological fracture from such a trivial cause as a blow from the fist of an infant. The roentgenogram taken at that time showed progressive atrophy of the mandible beyond the ordinary atrophy seen after extraction of all the teeth. The one important feature in the early roentgenographic picture was an abnormally large mandibular canal. No evidence of bone infection could be seen at that time. In twenty months time, the mandible was completely resorbed, leaving a flexible membrane in the place of the jaw.

During the four years following, the disease progressed further, involving various parts of the maxillary, palatine, and sphenoid bones. Salient facts of the case were the progressive nature of the disease; the involvement of the bones in the distribution of the fifth nerve with the persistence of a certain amount of pain in the distribution of the fifth nerve; the lack of evidence of hyperparathyroidism; the normal condition of all other bones; with increased calcium and vitamins in the diet, gain in weight has occurred, but the bone condition has been growing progressively worse; involvement of the bones in the distribution of the fifth nerve and its ganglia, together with persistent pain in these areas, suggests that the condition is a neurotrophic disorder.—*R. S. Bromer.*

SVENNINGSEN, O. K. Generalised xanthomatosis. *Acta radiol.*, 1933, *14*, 491–505.

The author reports a case of generalized xanthomatosis in a girl four years of age in whom the symptoms of the disease had begun at the age of two years. This disease is also known as dyspituitarism and Christian's syndrome. The three chief symptoms are pathological changes in the bones, particularly those of the skull, diabetes insipidus and exophthalmos. This child showed changes in almost all parts of the skeleton; there was considerable exophthalmos and the twenty-four hour urine was about 4,000 gm. The skull defects merged into large irregular areas and palpation of the head gave the sensation of feeling a flabby, badly blown-up rubber ball. The whole of the head was very tender to any contact. The first symptoms had been gingivitis, loosening of the teeth with intense pyorrhea and defects in the lower jaw bone. The Bence-Jones albumin reaction was negative. In spite of symptomatic treatment, quartz light baths and injections of pituitrin the child grew steadily worse and died two years after the beginning of the disease. Roentgenograms of the bones are shown.—*Audrey G. Morgan.*

PICK, LUDWIG. II. Niemann-Pick's disease and other forms of so-called xanthomatosis. *Am. J. M. Sc.*, May, 1933, *185*, 601–616.

In 1914 the Berlin pediatrician, Niemann, reported a case which he described under the title "An unknown disease picture." Pick subsequently developed the concept of that form of the essential internal xanthomatoses which is known in the literature as Niemann-Pick's disease.

The disease is a congenital, constitutional and familial one, affecting predominantly the female and with a predilection for the Jewish race. Enlargement of the liver and spleen is found in the first months of life. A rapid increase in the size of the abdomen, pallor and yellowish-brown discoloration of the skin, slight ascites, edema, bronchitis, and marked enlargement of the superficial lymph nodes are constant manifestations. The presence of large vacuoles in many lymphocytes and polymorphonuclear leucocytes is remarkable. The disease leads to death in all cases. Splenectomy or irradiation of the spleen offers only transitory improvement.

Complete diagnostic certainty is afforded by the demonstration of the characteristic cells filled with lipoid. This can be shown by puncturing the spleen or bone marrow. These cells have a definite and typical honeycomb or foam structure and one or two nuclei. The vacuoles lie most often at the periphery. The most striking finding at autopsy is the yellowish discoloration of the skin, the bone marrow, and all the viscera. It is an actual "xanthomatosis."

Since then, in Niemann-Pick's disease, cells of all the body tissues are involved, it appears necessary to explain the disease upon a single composite basis and not, for example, to limit

it to a primary insufficiency of the reticuloendothelial apparatus. Here, therefore, the pathogenesis revolves itself upon a primary metabolic disorder which leads to an overloading of the blood stream and the tissues with lipoids, especially with phosphatids. The foreign material in the blood stream and tissue fluids first of all mobilizes the entire histiocytic apparatus. In infancy the macrophage system is widespread and on this account the lipoid storage takes place in many other localizations after the natural storage localizations are overloaded. At this point the systematization ends. The excess amount of the substance present then forces the parenchymal cells, and even the cartilage cells, to store the substance. This results in extraordinary sites of localization and widespread parenchymal damage. The cause of this disturbance in metabolism is not understood.

Niemann-Pick's disease is compared with Gaucher's, Tay-Sachs', and Hand-Schüller-Christian's diseases and the group classified together as the generalized essential xanthomatoses.—*J. R. Andrews.*

Greene, Earle I., and Miller, Leo F. Isolated fratures of the os magnum and trapezium. *J. Bone & Joint Surg.*, July, 1933, *15*, 775–778.

Greene and Miller report 3 cases of fracture of the os magnum and 1 of fracture of the trapezium, with reproductions of the roentgenograms demonstrating these fractures. They conclude that isolated fractures of the carpal bones, except of the scaphoid, are uncommon. Every hand or wrist injury should have a most thorough study, including a critical roentgenographic examination. Many cases of so-called "sprains," "synovitis," "arthritis" and "rheumatism" are in reality unrecognized fractured carpal bones. Early recognition and adequate treatment are of extreme importance for the ultimate function of the wrist frequently depends upon this early knowledge. The isolated carpal fractures result more frequently from indirect trauma, although direct injury may be the factor. The prognosis in such fractures is usually good, since they heal by bony union.—*R. S. Bromer.*

Hermodsson, I. Zur Röntgenologie der Schultergelenkluxationen. (Roentgenology of luxations of the shoulder joint.) *Acta radiol.*, 1933, *14*, 275–282.

In this article the author studies a roentgen change in luxation of the shoulder joint that has heretofore been mistakenly interpreted in the roentgen literature. It is a loss of substance in the posterior lateral part of the head of the humerus above the greater tubercle. This has always been described as characteristic of habitual dislocation and this error is repeated in the latest edition of Schinz' Lehrbuch. It has been considered characteristic of habitual dislocation because it is never seen in the other shoulder or in other diseases of the shoulder joint. But no one seems to have thought of examining to see whether it occurs after a single luxation. The author has found only one report of such a finding. Didiée (*J. de radiol. et d'électrol.*, 1930) found the defect in 2 cases of primary dislocation and draws the conclusion that it must therefore have been a congenital defect.

The author discusses three groups of primary dislocations: axillary, subcoracoid and posterior, and illustrates them with roentgenograms. They show that this defect which has been considered characteristic of habitual dislocation occurs in first dislocations also. It is a primary bone injury—an infraction. The most pronounced form of this infraction occurs only in axillary dislocation. Similar but slighter bone injuries are sometimes seen in subcoracoid dislocation. In posterior dislocation similar bone injuries may occur in the region of the lesser tubercle. There is no evidence of any congenital or acquired change in the affected part of the head acting as a predisposing factor. The majority of habitual luxations are of the subcoracoid type and the bone injury is generally slight. The degree of injury of the bone therefore does not seem to be a factor in causing danger of recurrence. Injuries of the soft parts tend to produce recurrence by permitting greater mobility of the bone.—*Audrey G. Morgan.*

Lindsay, Merrill K., and Crosby, Edward H. Giant-cell tumor of the second cervical vertebra. *J. Bone & Joint Surg.*, July, 1933, *15*, 702–704.

A case of giant cell tumor of the spinous process of the second cervical vertebra is reported. The roentgen appearance was that of an expansile, cystic tumor involving the spinous and the transverse processes of the second cervical, the other vertebral bodies and interspaces being normal. The microscopic examination

showed the typical arrangement of fibrous connective tissue and giant cells. The occurrence of such a benign tumor in such location is unusual. The roentgenogram which is reproduced shows the lesion very clearly.—*R. S. Bromer.*

Greenberg, Lewis. Absence of the cervical spine; Klippel-Feil syndrome. *J. Bone & Joint Surg.*, April, 1933, *15*, 444–448.

Klippel and Feil in 1912, first described in detail the clinical, roentgenological and anatomical features of the relatively rare and obscure syndrome since known by their names. The most noticeable feature is an apparent absence of the neck; the head seems to rest directly on the trunk. Another feature is the unusually low level of the posterior hair line of the scalp; this actually reaches to the upper portion of the thoracic wall, posteriorly. Considerable limitation of the movements of the head in all directions is also present. With the above signs, frequently varying degrees of limitation of movements of the mouth, scoliosis or kyphoscoliosis of the upper dorsal vertebrae, elevation of the scapula, as in Sprengel's deformity, and other congenital malformations or deformities may be observed. Some patients exhibit mental retardation of varying degree, although others are of normal intelligence. A few have shown functional impairment of the upper extremities, suggestive of a common neurogenic origin. Roentgenologically there is a bony block of all or most of the cervical vertebrae, with malformation and irregular segmentation of the remaining fused vertebrae. The net result is usually a numerical reduction of its component elements, a bone defect in the posterior portion of this cervical block (spina bifida), and a thorax extending upward practically to the base of the skull.

Greenberg states that the condition is manifestly a developmental anomaly, but the precise chronological and morphological point of departure from the normal growth is still one of the mysteries of embryology. The total number of cases reported seems to be well below fifty. He reports briefly a case of a five year old white girl who presented the typical picture, clinically and roentgenologically, except that she had no restriction of the movements of the mouth.

Because of the deformity of the cervical spine it is often difficult to secure satisfactory roentgenograms. The importance in recognizing the condition lies not in the hope of remedying the deformity, but in its differentiation from two other conditions which present a somewhat similar appearance and which are amenable to treatment, namely, Pott's disease of the cervical spine and congenital torticollis. It is also desirable to investigate the cervical spine in all patients presenting Sprengel's deformity, since this is relatively frequently one of the elements of the Klippel-Feil syndrome.—*R. S. Bromer.*

Mitchell, C. Leslie. Isolated fractures of the articular processes of the lumbar vertebrae. *J. Bone & Joint Surg.*, 1933, *15*, 608–614.

Mitchell states that although fractures of the articular processes associated with severe fractures and fracture dislocations of the vertebrae are frequently seen and reported, very little mention has been made in the literature of an isolated fracture of this process. His series of 5 cases leads him to believe that the fracture is not so uncommon, that it is frequently unrecognized because of imperfect roentgenographic detail, and that the patient is often treated for low back strain. He believes that these isolated fractures result from direct violence. A severe force, causing the spine to be flexed both forward and laterally and at the same time rotated, produces impingement of the subjacent articular processes. The capsules, which are attached to the margins of the articular processes, are placed under considerable tension. If the force is sufficient, the combined action of impingement and capsular pull will produce a fracture of the tip of one or both of the subjacent processes.

An interesting feature observed in his cases was the persistent nonunion of the fragments with no attempt at callus formation. It is possible that incomplete immobilization of the fragments and the presence of synovial fluid may account for the failure of bony union.

If these lesions represented developmental anomalies with failure of union rather than actual fractures, Mitchell thinks one would expect to encounter these fragments quite frequently in routine roentgenograms of the spine. He has no knowledge, however, of their occurrence without a history of severe trauma. Three of his patients exhibited double lesions.

Treatment consisted of a short period of recumbency, followed by physiotherapy and the application of a support, such as a canvas belt or a Goldthwait back brace. For those cases that do not respond to this conservative treat-

ment, he advises operation, such as spinal fusion, arthrodesis of the involved joint and removal of the fractured fragments.—*R. S. Bromer.*

Nichols, B. H., and Shiflett, E. L. Ununited anomalous epiphyses of the inferior articular processes of the lumbar vertebrae. *J. Bone & Joint Surg.*, July, 1933, *15*, 591–600.

The authors state that, so far as they know, an ununited epiphysis of the inferior articular processes of the lumbar vertebrae has not been previously described. Attention has been directed to its importance on account of the similarity to cases of fracture as described by Koch. In a review of the literature no description of such an epiphysis could be found. They report in detail 7 cases of anomalous formation of the inferior articular processes of the lumbar vertebrae. These were discovered during routine examinations of the lumbosacral region and of the genitourinary tract. Most of the examinations were made because of non-radiating pain in the lower back which increased with exertion. Two patients gave a history of trauma but in most patients the back pain was not associated with trauma. Two men in the group had chronic prostatitis; 3 were laborers doing heavy work requiring some lifting; 1 was a physician whose chief disability was hypertensive heart disease; and there was 1 woman whose chief complaint was chronic constipation and lumbago. The ages of the men varied from twenty-five to fifty-seven years; the woman was forty years of age.

Stereoroentgenograms of the spine were made in all the cases, this being found superior to examination by single anteroposterior and lateral roentgenograms. This apparent anomaly has the characteristic appearance of an ununited epiphysis. The linear defect extends from above downward and medially, or it may be almost horizontal, bisecting the inferior articular process. The defect may extend through the facet face, or just above the base of the lamina. Each smooth edge is bordered by an apparently increased bone density as is observed in ununited epiphyses. In their experience the condition has not been observed as involving the superior articular process. When one becomes familiar with the typical roentgenographic appearance, the condition offers no diagnostic difficulties.

Nichols and Shiflett believe this condition is an ununited epiphysis, probably anomalous, of the inferior articular process of the lumbar vertebrae. Its appearance may be misinterpreted as a fracture. Embryological confirmation that this is an ununited epiphysis is lacking. They assume it is logical to believe that the anomaly is not responsible for the symptoms which were present in their cases.—*R. S. Bromer.*

Williams, Paul C., and Yglesias, Luis. Lumbosacral facetectomy for post-fusion persistent sciatica. *J. Bone & Joint Surg.*, July, 1933, *15*, 579–590.

In the authors' clinic, 74 per cent of the cases suffering with sciatica due to pathological bone or joint changes showed a characteristic lesion which consisted of a narrowing or a complete loss of the lumbosacral intervertebral disc. This in turn had caused a subluxation of the joints formed by the articular facets of the fifth lumbar and first sacral vertebral bodies, a constriction of the foramina formed by these segments, and an arthritic lipping of the adjacent surfaces of these two bodies. This had resulted from the abnormal contact and stress following the loss of the disc.

From a roentgenographic standpoint these patients are divided into two groups: first, those whose roentgenograms show a complete loss of the disc; second, those whose roentgenograms show a narrowing of the lumbosacral disc only posteriorly. In the second case, the roentgenographic finding is a very frequent and common lesion, and because of this no significance has been attached to it. However, in the authors' experience, every patient in whom this roentgenographic change has been found, gave a history of pain in the lower part of the back and down one or both extremities, which usually occurred in attacks. Symptoms were not continuous. It is therefore possible to find the lesion in patients who are not complaining of the characteristic symptoms and who do not present the usual clinical findings. Not all cases of sciatica are due to pathological bone or joint changes nor are all cases due to the loss of the lumbosacral disc. Every case should have a thorough neurological examination and the usual search for any toxic chemical or mechanical irritation. Lumbosacral anomalies are, however, found in a great many patients. This is especially true in those cases showing a unilateral sacralization of the fifth lumbar vertebra and in those showing a lumbarization of the first sacral vertebra. Williams and Yglesasi

believe that irritation of the nerve root in this group is due to practically the same mechanical alteration as in those cases showing a loss of the lumbosacral disc.

A point of differentiation is made between sciatic neuralgia and neuritis. Neurologists make a diagnosis of sciatic neuritis when there are no definite motor or sensory changes, while the term neuralgia is applied to those cases which have pain without neurological changes along the course and distribution of the sciatic nerve. It is the authors' opinion that most cases start with a neuralgia and as irritation increases in severity and the clinical course becomes more prolonged there may be both motor and sensory changes, so that the end stage of a severe case presents a sciatic neuritis. The pain in sciatic neuralgia and the nerve changes in a neuritis are usually confined to the distribution of the fifth lumbar nerve segment.

Complete fixation of the lumbosacral joint by a plaster spica for a period of twelve to fifteen days will give at least temporary relief of sciatic pain in the greater percentage of cases. In a similar manner a lumbosacral fusion, by removing the factor of motion, will give permanent relief in the majority of cases suffering from pain in the lower back and sciatic irritation. A lumbosacral facetectomy, after fusion had been accomplished, gave complete relief of symptoms in the one case in their series which failed to get relief from a lumbosacral fusion.—*R. S. Bromer.*

Moffat, Barclay W. Enlargement of the intervertebral disc associated with decalcification of the vertebral body: A compensatory hypertrophy. *J. Bone & Joint Surg.*, July, 1933, *15*, 679–695.

Moffat states that it has been noticed by all orthopedic surgeons that a roentgenogram of the spine will occasionally show an increase in height of the intervertebral disc, this structure assuming a biconvex form. The significance of this change and the mechanism by which it comes about have not been investigated.

He reports 5 cases in which these changes in the shape of the intervertebral disc were very marked and were accompanied by decalcification of the vertebral bodies. These patients all came complaining of backache, following slight trauma in 4 cases, and none in the other. Besides the above changes, by roentgen examination, there was found to be a fracture of one or more of the vertebral bodies as the source of the pain of which the patient complained. It is logical, Moffat assumes, to consider that these fractures were associated with the underlying condition in the spine which led to the decalcification and to the increase in size of the intervertebral disc. In the absence of any local pathological process such as osteitis or neoplasm, a pathological fracture of the spine may take place through faulty metabolic processes.

Moffat thinks that the process seems to be one of osteoporosis of the bodies with secondary changes in the intervertebral discs, and that obviously the changes in the body were due to replacement of the normal bony tissue by malignant cells, or to decalcification. The assumption of the spherical form by the disc has been attributed by Schmorl and others to expansion of the nucleus pulposus, due to release of pressure, the result of softening of the adjacent bodies. As a cause of the latter condition Schmorl suggests chronic disease. Moffat believes that the process is one of compensatory hypertrophy of the cellular elements of the discs rather than an expansion of the nucleus pulposus.—*R. S. Bromer.*

Mossessian, Zaré. Un cas d'hémangiome de la colonne vertébrale. (A case of hemangioma of the vertebral column.) *J. de radiol. et d'électrol.*, July, 1933, *17*, 363–371.

The author describes in detail a case of hemangioma of the vertebral column which occurred in a woman fifty-eight years of age. The first symptoms were those of sciatic neuralgia and the patient was treated as such. However, since there was no improvement after four or five months of physiotherapy, later further investigations were made. At this time the patient was seen by the author who found on roentgen examination a modification of the structure of several vertebrae consisting in a reticulated rarefaction with enlargement of the contours and signs of deforming spondylosis. A diagnosis of hemangioma of the vertebrae was made and operation was advised. However, since the patient refused to consider such a procedure radiation therapy was undertaken with satisfactory symptomatic alleviation lasting now for over two years.

In connection with this case the author has reviewed the literature and was able to collect a total of 18 cases of hemangioma of the vertebral column which are arranged in tabular form. A bibliography of 21 articles is also included.—*T. Leucutia.*

Harding, D. B., and Garr, Chas. C. Blastomycosis of bone. *South. M. J.*, April, 1933, *26*, 315–320.

Bone involvement in systemic blastomycosis is frequent, usually affecting those of the extremities. The primary bone lesions are almost certainly hematogenous in origin, with an unknown portal of entry. Pain, of sudden onset, worse at night, is a constant early symptom. There is little or no elevation of temperature, unless the bone involvement is part of a systemic infection. Resembling acute osteomyelitis at the onset, the only differentiating features are the mild febrile reaction and the absence of a leucocytosis. Without characteristic cutaneous lesions, there is no hint as to the type of the infection. Later, the persistent sinuses, following spontaneous or surgical drainage, together with the more or less characteristic roentgen appearances, suggest a search for the blastomyces. A chronic course suggests tuberculosis and the presence of a positive Wassermann may serve only to confuse the diagnosis. Coccidioidal granuloma can be differentiated only by laboratory methods. There is one constant feature of the variable roentgen findings; the presence of a sharp line of demarcation between the involved areas and the adjacent normal appearing bone. In the short bones, single or multiple areas of complete destruction are seen, involving chiefly the central portions of the bones. If close to the articular surfaces, a thin shell of bone may remain beneath the cartilage, or the joint may be entirely destroyed. With extension into the articulating bone, marked osteoporosis may cause difficulty in determining the exact extent of involvement. Early lesions in the long bones may show periostitis or central necrosis, alone or in combination. Multiple adjacent foci usually coalesce. For the establishment of the diagnosis, the possibility of the lesion must be considered, since the organisms may be readily found by examination of pus from the infected tissues. Ten cases are reported in detail with illustrative roentgenograms.—*W. R. Brooksher.*

Torelli, Gastone. Contributo allo studio della artropatie tabetiche. (Tabetic arthropathy.) *Radiol. med.*, 1933, *20*, 919–928.

A case of tabetic arthropathy in a man aged forty-nine is described and illustrated with roentgenograms. The patient had very few clinical signs of tabes. The question of the relationship between tabetic arthropathy and arthritis deformans has been discussed a great deal. The author concludes from a study of the literature and of his own case that tabetic arthropathy is only an arthritis deformans that has taken on special characteristics because it has developed in a patient with tabes. It may be a very early symptoms of tabes.—*Audrey G. Morgan.*

Perotti, Desiderio. Distrofia metaepifisaria congenita generalizzata (osteocondrodistrofia). (Congenital generalized meta-epiphyseal dystrophy—osteochondrodystrophy.) *Radiol. med.*, Sept., 1933, *20*, 1183–1193.

Brailsford gave the name of osteochondrodystrophy to a new familial bone disease first described by Morquio in 1929. The author describes a case of it in a girl aged five and a half. Her growth was retarded and at the age of nine months her parents noticed a beginning kyphosis which increased as she grew older, and coxa vara and genu valgum also developed. The child's body was dwarfed but her limbs were normal in size. Her head was large, suggesting hydrocephalus, her chest bell-shaped and her psychic development was retarded. The bodies of the vertebrae were flattened and increased in length and breadth, as if they had been crushed by the weight of the body and had consequently spread horizontally. The Wassermann reaction was negative in both the child and her parents. Roentgen examination showed retardation and irregularity in the ossification of cartilage due to insufficient apposition of calcium salts, resulting in deformity of the bones from weight bearing. There was also diffuse hyperchondroplasia with blocks of cartilage included in the metaphyses of the bones.

This disease differs from achondroplasia in the shortness of the trunk with normal length of the limbs and from both that disease and infantile meta-epiphyseal dystrophy in being a systemic disease involving the whole skeleton and being familial and associated with psychic defect.

The author believes it is an independent disease caused by endocrine disturbances originating in intra-uterine life. Changes in the sella turcica indicated disturbance of the function of the hypophysis; the costal hyperchondroplasia and enlargement of the upper mediastinal shadow indicated hypertrophy of the thymus and the psychic defect, bradycardia and irregular appearance of the centers of ossification

indicated involvement of the thyroid. The parathyroids have an influence on calcium metabolism and they also were probably affected.—*Audrey G. Morgan.*

WISHNER, JOSEPH G. Chronic sclerosing osteomyelitis (Garré). *J. Bone & Joint Surg.*, July, 1933, *15*, 723–732.

This type of bone infection occurs frequently enough to cause some difficulty in diagnosis and treatment. The most likely cause is bacterial and trauma is usually the exciting factor. The difference between this condition and acute osteomyelitis is apparently one of degree. In 3 of the 5 reported cases the staphylococcus was isolated. The so-called "silent foci" of Phemister are mild foci of infection which have healed. They may be reactivated by trauma and so cause symptoms.

From a pathological standpoint there are two types of cases. In one, a true osteomyelitis, the marrow is first involved. It becomes cellular, shows an increased fibrosis, the cells of this fibroid tissue undergo metaplasia and become bone corpuscles. The marrow cavity is entirely obliterated. In the second type the cortex is first invaded, the process starting as osteitis. In this type there is increased osteoblastic activity, so that the lamellae undergo progressive thickening and the marrow spaces become correspondingly reduced. The cortex becomes thickened, and the medullary cavity is diminished, but not obliterated. This bone is usually dense and like ivory, commonly described as eburnated bone. In this type some bone lengthening may occur. The symptoms are variable.

The roentgenogram is the one important aid in diagnosis but Wishner states that it helps only in late cases. The clinical laboratory is of little help. There is usually slight elevation in the leucocyte count. The Wassermann reaction is important.

Clinically the reported cases fall into two groups. In the first the involvement is in the shaft of a bone, the tibia, or femur. The symptoms are relatively mild, merely local pain, slight disability and the diagnosis is comparatively simple. In the second group the area involved is near a joint. In this group the symptoms are more marked, the disability is more severe and the diagnosis is difficult.

The treatment is operative. It is comparable to incision and drainage in other infections. Simple excision of a section of bone gives relief of symptoms and return of joint function, except where contractures are of long standing. Incision into bone apparently allows for a lessening of tension and reëstablishment of circulation. In the second group treatment by drainage of the bone produced marked improvement.—*R. S. Bromer.*

PAPADOPOULOS, ALEXANDER S. Ewing's tumor; an unusual case. *J. Bone & Joint Surg.*, July, 1933, *15*, 789–792.

The author records the first case in Greece of an endothelioma of bone. His case is of interest because the first roentgen examination showed a clearly circumscribed area of condensive osteitis in the upper third of the tibia, such as is never seen in tuberculosis although the history for several weeks previous had suggested this condition. One month after the first, a second roentgenogram was made and this showed a longitudinal and irregular extension of the dark shadow previously found, forming a column an inch in length with a zone of rarefied bony tissue on the outer side. The periosteum did not seem to be involved in the local process. Clinical signs recorded at that time were atrophy of the thigh, two-thirds of an inch, local swelling and elevation of the temperature, with the appearance of dilated subcutaneous veins. Operation was later performed and roentgenograms six weeks after showed marked extension of the lesion with destructive process greater than bone condensation. Histological examination proved it to be composed of a small round cell sarcomatous structure, with a peritheliomatous character. It also revealed the local anatomical characteristics of Ewing's sarcoma in which only blood and small particles were found which were considered to be the result of ordinary inflammatory reaction. These anatomical irregularities are frequently mentioned in the literature of Ewing's sarcoma.—*R. S. Bromer.*